

Utah Science

Volume 45 | Number 4

Article 1

12-1984

Utah Science Vol. 45 No. 4, Winter 1984

Follow this and additional works at: <https://digitalcommons.usu.edu/utscience>

Utah Science is produced by Utah State University Agricultural Experiment Station.

Recommended Citation

(1984) "Utah Science Vol. 45 No. 4, Winter 1984," *Utah Science*: Vol. 45 : No. 4 , Article 1.

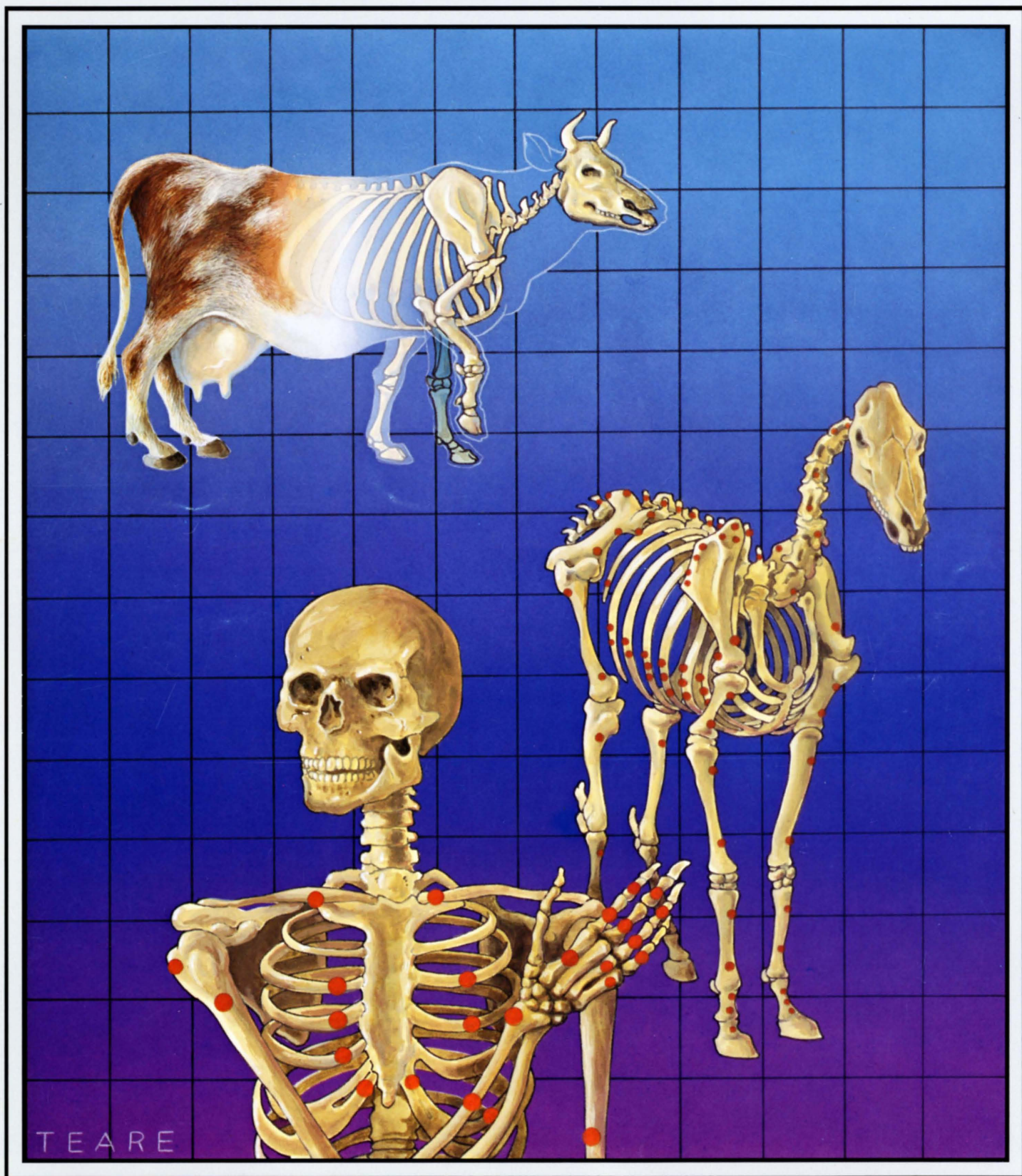
Available at: <https://digitalcommons.usu.edu/utscience/vol45/iss4/1>

This Article is brought to you for free and open access by the Journals at DigitalCommons@USU. It has been accepted for inclusion in Utah Science by an authorized administrator of DigitalCommons@USU. For more information, please contact digitalcommons@usu.edu.



UTAH SCIENCE

UTAH AGRICULTURAL EXPERIMENT STATION WINTER 1984 VOLUME 45 NUMBER 4



UTAH SCIENCE

UTAH AGRICULTURAL EXPERIMENT STATION

97 PHOSPHORUS NEEDS OF BEEF CATTLE

J. W. Call, J. E. Butcher, J. L. Shupe, A. E. Olson, and
J. T. Blake

Levels of phosphorus, suggested for normal growth and reproduction, are outlined. Symptoms and lesions of phosphorus deficiency in beef cattle are discussed.

100 FAT CATTLE

D. Bailey and B. W. Brorsen

The price differences by month and year between Utah and other regional markets for fat cattle were charted and analyzed to help the producer to make effective marketing decisions.

104 THE MALNUTRITION/DIARRHEA CONNECTION

R. W. Sidwell, B. B. Barnett, and A. W. Mahoney

Using pregnant and newborn mice, researchers have reconfirmed the value of a well-balanced diet, particularly in the expectant mother. Their emphasis was on the connection between dietary adequacy in the pregnant mother and rotavirus-induced diarrhea in the offspring.

108 COMPARABLE BONE DEFECTS

J. L. Shupe, A. E. Olson, N. C. Leone, E. J. Gardner and Y. Elsner

Several musculo-skeletal disorders of livestock have been studied at USU including fluoride toxicosis and hereditary multiple exostoses (HME) in both animals and humans. The results of the experiments on the animals have had valuable applications to humans.

112 A FEED SOURCE OF THE FUTURE

D. C. Dobson, N. J. Stenquist and J. O. Anderson

Composted fresh cage layer waste processed into a dry product form would be suitable as a feed ingredient for animals.

116 PANTOTHENIC ACID

W. O. Song, B. W. Wyse, and R. G. Hansen

Due to lack of standardized methods available for determining pantothenic acid in human beings, results from different laboratories, have been inconsistent until now. Research at USU encompasses the status of pantothenic acid in the diet of the elderly, pregnant and lactating women, and adolescents in Utah, a study to be completed by summer 1985.

120 MUTTON IN SUMMER SAUSAGE

D. T. Bartholomew, B. L. Woodbury, and C. I. Osuala

Summer sausage is usually made from all beef ingredients but experiments at USU show that combining mutton meat with beef fat can produce a highly desirable sausage without any mutton off-flavor.

124 *IN VITRO* TOXICITY EVALUATION

R. P. Sharma and M. Yoneyama

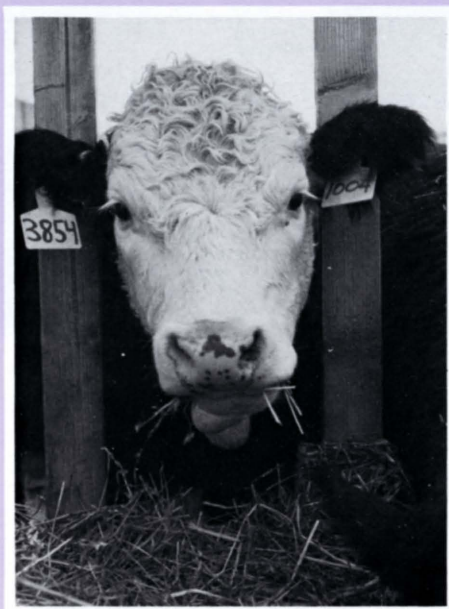
To predict toxic responses in human health hazards, data have been developed from experiments on tissue and cell culture systems and evaluated to assess the toxicity of chemical exposure in animals and humans.

132 INDEX of PUBLICATIONS 1984

133 LIST of NEW PUBLICATIONS 1984

Cover Illustration
BRADLEY TEARE

PHOSPHORUS NEEDS OF BEEF CATTLE



Phosphorus is considered the mineral nutrient most often deficient in beef cattle. The deficiency has been claimed to be widespread. Programs of supplementing phosphorus to livestock in excess of recommended levels, e.g., National Research Council (NRC), have been advocated. Some people claim increased fertility, promotion of growth, and improved general herd health if animals are supplemented with phosphorus. Others report reduced fertility, decreased growth, and impaired health with deficiencies of phosphorus. Some researchers report no benefit from supplementation. This research was undertaken at Utah State University (USU) to determine levels of phosphorus needed for normal reproduction and growth, and to describe symptoms and lesions of phosphorus deficiency in beef cattle.

Experimental Procedures

Hereford heifers were individually fed two times a day from weaning (200 days of age) through their 8th gestation with dietary phosphorus (P) levels being the only treatment variable. All animals received the same basal feed with free access to a phosphorus-free mixture of salt, trace minerals, and vitamins. The basal diet consisted of various combinations of pelleted or chopped alfalfa, wheat straw, grass hay, pelleted sugar beet pulp, and beet molasses. The lowest level of dietary phosphorus in any time period was from the natural feed sources. The higher phosphorus (P) levels were obtained by adding monosodium phosphate (MSP). From the first through the fourth gestation, the 24 low (L) P cows received 6 to 13 grams P per day and the 24 high (H) P cows received 21 to 40 grams P per day (Figure 1). The total phosphorus intake

was increased as the heifers grew. Feed intake (Figure 2), body weights (Figure 3), and pregnancy rate (Figure 4) were not different between treatments during these four gestations. At the end of 4 gestations, 42 cows remained on the project (6 deaths were unrelated to treatment) and 1/2 of (H) cows and 1/2 of (L) cows were put on a very low < (HI or LI) > diet, with basal feed providing less than 7 g P/day (Figure 1). The cows on the high P level received approximately 35 g P/day and cows on the low P level received approximately 9 g P/day (Figure 1).

Results and Discussion

Most of the cows that received less than 7 g P/day for a year or more developed an abnormal complex syndrome. The clinical signs and lesions included general unthriftiness, body weight loss, reduced feed consumption, lameness, abnormal stance, impaired reproductive performance, recumbency, and finally emaciation and death (3 of 21 died) (Figures 2, 3, 4, 5, and 6). There was no apparent difference in clinical response between the cows shifted from (H) P to (HI) P when compared to those shifted from (L) P to (LI) P.

The cows receiving approximately 9 or 35 g per day during the same period did not develop adverse clinical signs.

The feeding of less than 7 g P/day began in November, 1977 (Figure 1). All cows on this level calved normally in April, 1978, and were rebred in July and August of 1978. An apparently normal calving in April, 1979, was followed by failure to rebreed (19 of 21 cows) in July and August, 1979 (Figure 4). During the period between November, 1977, to November, 1979, the cows on high and low P had no significant difference in calving performance (Figure 4).

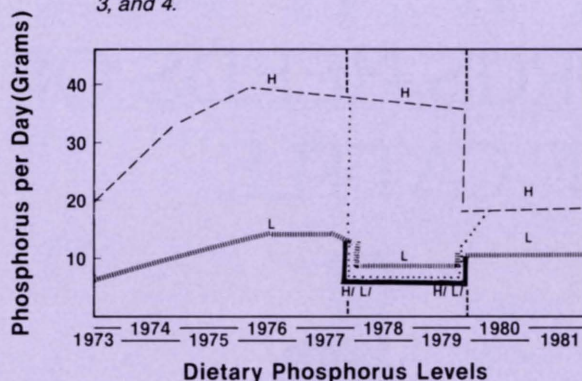
During November, 1979, the P levels were readjusted. The high P level was reduced from the approximate 35 g per day to 19 g per day (approximate NRC recommended level). The low P level was raised to approximately 12 g/day. The animals on the very low P levels were returned to their respective high and low groups (Figure 1).

After a month on the higher levels of phosphorus, the stressed animals had regained much of their voluntary feed consumption, and were clinically more normal in appearance. After approximately two months of recovery (February, 1980), bulls were introduced to these previously stressed cows and they bred successfully within two breeding cycles (42 days) (Figure 4).

Blood phosphorus levels tended to be depressed in the very low < (HI and LI) > cows.

After 14 to 24 months on the less than 7 g P/day feeding, several animals on each treatment (HI) and (LI) were necropsied for morphological and analytical evaluation. Severe bone changes occurred in the (HI) and (LI) P cows (Figure 5). The low (L) P cows, receiving approximately 9 grams per day for 14 to 24 months, did

FIGURE 1. Phosphorus in g/day fed with no variation for gestation, lactation maintenance. Note the extra stress period Nov. 1977 to Nov. 1979—the same time scale is on Figures 1, 2, 3, and 4.



LEGEND

--- H -High
 L -Low
 HI -Very Low
 — LI -Very Low

FIGURE 2. Body weights were not affected until after severe stress (very low P). The weights returned to normal after refeeding began in late 1979.

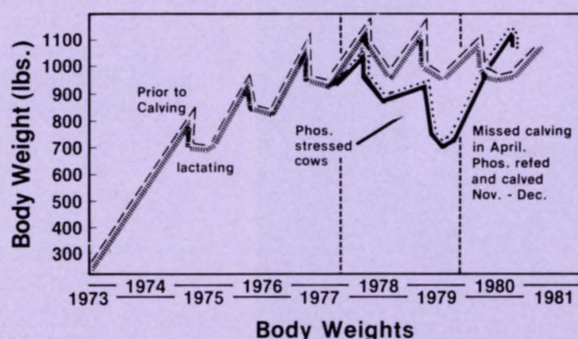
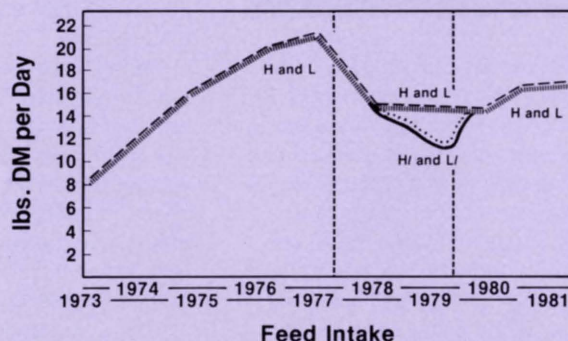
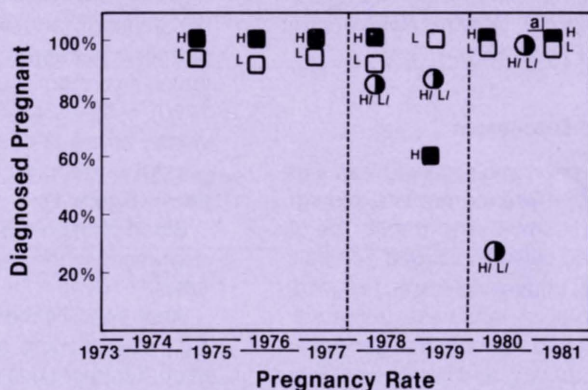


FIGURE 3. After approximately a year, the lowest P resulted in depressed feed intake resulting in total nutrient deficiency.



LEGEND

■ H-21 to 40 g P/day
 □ L-6 to 13 g P/day
 ● HI, LI-less than 7 g P/day



a) Open cows (1980), pregnant after increased phosphorus

FIGURE 4. Reproduction was seriously impaired after the HI or LI diets (less than 7 g P/day) were imposed for approximately 18 months.



FIGURE 5. The bone depletion is serious on the extended feeding of very low phosphorus.

have subtle bone changes, although reproduction was not impaired. The degree of bone changes was related to the level of phosphorus intake and time on very low (HI) and (LI) P. Bone changes preceded detectable clinical signs. These changes included various degrees of osteoporosis (loss of bone mass). Some severely affected animals had spontaneous or secondary fractures. Some of the fractures showed various degrees of poorly mineralized healing formations. Bone became thinner, more porous, and walls became thinner (Figure 5).

The syndrome associated with phosphorus deficiency in beef cattle on this experiment developed in an insidious manner, with an overlapping of signs and lesions among groups. One of the primary signs of P deficiency was reduced feed consumption, which compounded the problem with secondary responses (Figures 6 and 7).

Cows adapt to a very wide range of phosphorus intake ranging from natural feed sources to heavy supplementation with MSP and to a very low phosphorus intake for an extended length of time. Phosphorus-depleted beef cows responded quickly to higher levels of phosphorus, and regained their reproductive ability and apparent good health.

The question is not whether a cow needs phosphorus, but how much? The levels for a 1,000 pound cow range from 12 g P per day for early gestation to 27 g for average milking ability (lactation). This would average to 17.5 g/day over the year. This research indicated that the



FIGURE 6. Some emaciated cows produced apparently normal calves.



FIGURE 7. The low phosphorus, 9 g/day, did not cause emaciation.

range of intake with time may be logical, but not necessary, since the bone is effective as a phosphorus reservoir or buffer.

A comparison of recommended dietary phosphorus level and the levels fed are illustrated in Table 1. It should be noted that the recommended level is a weighted average of phosphorus recommended at various levels throughout the year and this experiment had animals fed at a relatively constant daily level during the time periods indicated in Figure 1.

With this data it has been demonstrated that:

1. Mature producing cows may maintain normal (high) reproductive performance on 50 to 70 percent of the commonly recommended levels of dietary phosphorus, if other nutrients are maintained at adequate levels.
2. The 40 percent level (7 g/day for 1,000 lb. cow) for 14 to 24 months will result in loss of bone mass, emaciation, rough hair coat and an abnormal clinical appearance of high withers. Loss of body weight and voluntary reduction of feed intake lead to a complicated nutrient deficiency with final result of reproductive failure and, in some cases, death (3 of 21 died). Dramatic improvements, however, were observed by increasing phosphorus in the diets of these cows. The clinical appearance improved within days, the voluntary feed intake increased within a month and rebreeding was successful within two to three months after

increasing the 7 g P/day to 12 or 17.5 g P/day.

Cost Factor

Phosphorus feeding beyond the needs of the animal does have a cost. One of the cheaper common dietary supplements for phosphorus costs \$19.50 per hundred pounds with 18 percent phosphorus. This results in one (1) gram of phosphorus per cow per day per year being worth \$.85. If the recommendation is 17.5 grams per day and 12 grams per day is adequate, then 17.5 minus 12 equals 5.5 grams per day potentially in excess of production needs. This would be 5.5 times \$.85, or \$4.68 per cow per year. This example will vary with different conditions, but it would be a potentially costly "safety factor." The most costly condition would occur when cows receive the commonly recommended levels of dietary phosphorus from natural sources and have supplemental phosphorus added.

This research indicates that a high level of phosphorus would not be detrimental, just costly.

Another way to approach the cost is to use phosphorus as a soil amendment, which is commonly done. If the soil phosphorus fertilization results in increased crop yields, as is expected, especially for legume crops on land that has low soil phosphorus, the increased crop yield may pay for the phosphorus fertilization and the animals have the benefits of more crop production and higher concentration of phosphorus in the forage. The cattle on

this experiment were efficient in using the phosphorus from the forage sources. This chain supplementation of phosphorus to the soil, the plant, and eventually the animal is a well-established procedure.

TABLE 1. Approximate recommendations of phosphorus intake, for 1000 lb. beef cows as compared to the USU experiment.

Recommended	Level fed on experiment	% of Recommended
g/day	g/day	%
17.5	35 (high to 1979)	200
17.5	12 (low)	69
17.5	9 (low 1977-79)	51
17.5	7 (very low 1977-79)	40

ABOUT THE AUTHORS

Jay W. Call is a professor of clinical medicine and reproductive physiology in the Department of Animal, Dairy and Veterinary Science.

John E. Butcher is a professor of nutrition and management of cattle and sheep in the Department of Animal, Dairy and Veterinary Science.

James L. Shupe is a professor of pathology and toxicology in the Department of Animal, Dairy and Veterinary Science.

Arland E. Olson is a research associate of histotechnology in the Department of Animal, Dairy and Veterinary Science.

Joseph T. Blake is a professor emeritus. His primary research was in nutrition and physiology.

FAT CATTLE

REGIONAL PRICE DIFFERENCES 1978-1983

FIGURE 1. Mean price differences between Utah fat cattle prices and other regional markets by month, 1978-83.

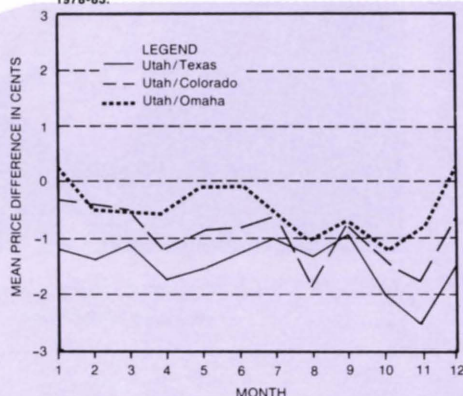


FIGURE 2. Seasonal variation in Utah fat cattle prices, 1978-83.

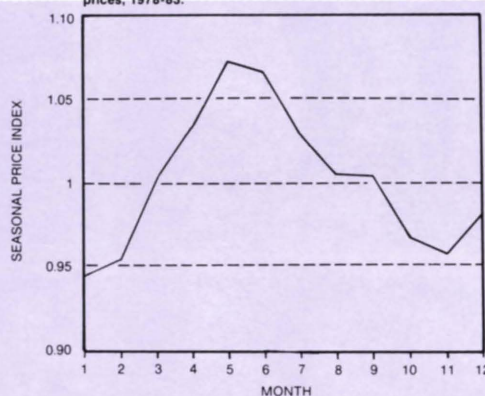


FIGURE 3. Seasonal variation in Colorado fat cattle prices, 1978-83.

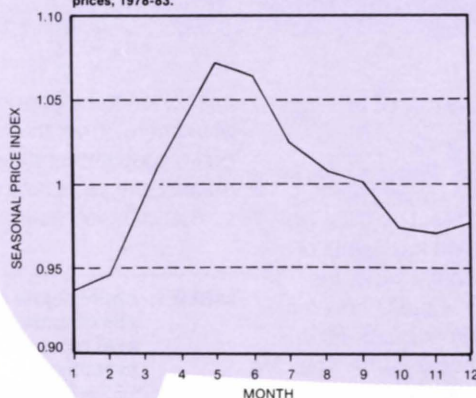
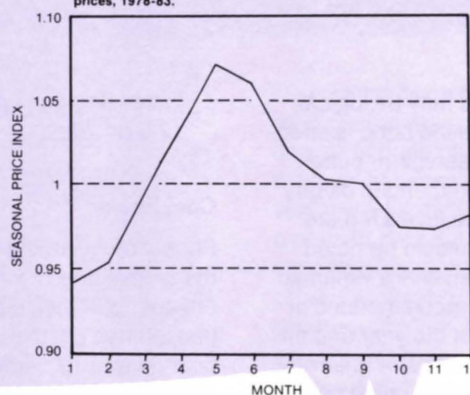


FIGURE 4. Seasonal variation in Texas fat cattle prices, 1978-83.



D. BAILEY and B. W. BRORSEN

Producers and researchers are concerned that Utah cattle prices are lower than other areas of the country (Snyder, 1983). There is also an increased interest in market information in Utah as evidenced by the new state-wide market information system developed by the State Department of Agriculture and Extension Service. Identifying the price relationships between Utah and other regional markets should be useful to both producers and buyers of Utah fat cattle.

Equilibrium prices for different locations are determined by supply and demand conditions. The process of reaching an equilibrium is termed price discovery. Arbitration processes force prices to an equilibrium across space. In the case of regional prices, the arbitration takes the form of transportation activities. If these activities are performed efficiently, then the price difference between two locations will be less than or equal to the transportation costs.

This article investigates price relationships between Utah and other regional markets. First, price differentials and their seasonal variations among four markets are compared. Second, Utah price differentials relative to other regions are compared to transportation costs to determine if short-run disequilibriums do exist.

Structure of Utah's Beef Packing Industry

In 1982, approximately 220,900 head of cattle (excluding calves) were slaughtered in federally inspected and other slaughter plants in Utah (Utah State Department of Agriculture). Of this number, approximately 207,240 head were slaughtered in the four largest packing plants in the State. This yields a four-firm concentration ratio (CR_4) of about 94 percent. Idaho's CR_4 for cattle slaughtered in federally inspected plants was approximately 77 percent. Virtually all of this activity

occurred in the Southern Idaho area (Rutts, 1983).

Since packers in Utah and Southern Idaho compete to buy fat cattle, the relevant market area for Utah stretches the length of Utah, reaches into about the Boise area of Idaho, and then across Idaho to Idaho Falls (Andersen et al., 1983). The packer CR_4 for all cattle slaughtered in this market area in 1982 was 73 percent.

If only fat cattle slaughter numbers are considered, the concentration ratios increase considerably. The four-firm concentration ratios for commercial-slaughter, fat cattle in Utah and Idaho during 1982 were over 99 percent and 94 percent respectively. The CR_4 for the combined Utah and Southern Idaho area in 1982 was 89 percent for fat cattle.

These concentration ratios would be considered high in almost any industrial setting. Questions pertaining to the amount of influence any particular

packer(s) may exercise on prices may be relevant based on the high concentration of the market. High concentration ratios do not necessarily, however, indicate that meat packers depress prices in any particular area (Ward, 1979). Regional markets remain in equilibrium so long as excess profits to packers do not exceed shipment costs to other regions. The test of market efficiency rests upon the ability of the market price to accurately reflect the market and to correctly react to changes in market conditions.

Data and Modeling Procedure

Utah prices were analyzed relative to those of three other markets. The markets considered besides the Utah-Eastern Nevada-Southern Idaho (Utah) market are the Texas Panhandle (Texas), Omaha, and Colorado-Kansas (Colorado). The data consisted of the weekly quoted prices for fat cattle graded choice (yield grade 2-4) for the four separate markets between January 1, 1978, and June 4, 1983. These data are published in *Livestock, Meat, Wool Market News* (USDA), with the exception of the Utah-Eastern Nevada-Southern Idaho prices, which were obtained from the Utah State Department of Agriculture. The data were analyzed by

- 1) determining the actual behavior of prices and differences for the four regions (i.e., seasonal behavior and magnitudes of price differences),
- 2) analyzing transportation costs to determine if they are less than the average price differentials.

Price Differentials

Seasonal price movements refer to regular and systematic price movements within a year. The period(s) of increases (decreases) in price would be expected to occur either when supplies are short (plentiful) or demand increases (decreases). Cattle prices are expected to be highest in the spring when fewer fat cattle are coming out of feedlots, and lowest in the fall when fat cattle are coming out of feedlots in relatively large numbers.

Perishable commodities generally have larger seasonal price variations than storable crops. Strawberries, for instance, are difficult to withhold from the market during periods of heavy shipment (Shepherd and Futrell, 1982). This leads to low prices for several months until supplies lessen and prices rise. Fat cattle may not be profitable withheld from slaughter once they

reach a desirable grade. This makes orderly marketing more difficult during peak marketing periods (e.g., fall). Generally, the tendency toward market equilibrium dictates that price differences between regional markets may not exceed transportation costs. Otherwise, commodities will move between regions until the markets come back into equilibrium.

Charting was used to analyze seasonality in fat cattle prices and price difference between the four regions. The results may indicate the periods of the year when shipment of cattle from Utah to other regions is most likely to be advantageous. The data were also tested for any evidence of inefficiencies across regions in the form of price differences greater than transportation costs plus a normal return.

Shipment Costs

Total costs for shipping fat cattle were assumed to be transportation (trucking) plus shrinkage costs. Transportation costs were estimated using the quoted price on July 28, 1983, from Logan, Utah. These trucking costs were quoted* as \$2.10/cwt., \$2.30/cwt., and \$1.50/cwt. to Amarillo, Texas; Omaha, Nebraska; and Denver, Colorado, respectively (Miller, 1983). These costs were then deflated using a standard transportation cost index to estimate the mean costs for 1978-82 (Table 1) (USDA ERS and U.S. Department of Commerce).

An Iowa State University study estimated shrinkage at approximately 0.61 percent for each 100 miles in transit (Minish, 1982). About half of this shrinkage is excretory and half tissue loss. Shrinkage costs vary according to the elapsed time between when cattle arrive at their destination and when they are weighed. If cattle are permitted to eat and drink before being weighed, a large portion of the excretory shrink will be recovered. Tissue loss, however, may take up to 16 days and more to replace (Minish, 1982).

The distances from Logan, Utah, to Denver, Amarillo, and Omaha are approximately 500, 900, and 1,000 miles, respectively. This implies an average shrink of between 3.05 percent and 6.1 percent from Logan to Omaha depending upon if the cattle are fed and watered before weighing. A shrinkage of between 2.7 percent and 5.49 percent would be experienced between Logan and Amarillo, while

1.5 percent to 3.05 percent shrinkage would be realized on shipments to Denver.

The cost of shrinkage may be calculated as percent shrinkage \times destination price. A fat steer priced at \$65/cwt, with 6.00 percent shrink would be worth $65.00 \times .94 = \$61.10$. Thus, the shrink loss (cost) would be \$3.90/cwt. Total costs of shipment were calculated for both full shrink and half shrink (excluding excretory shrink).

Regional markets in equilibrium will see no price differences between regions that exceed shipment costs, otherwise shipment would take place between regions until the regions come back into equilibrium. Some short-term disequilibria may occur in a market and allow profitable interregional shipments of commodities. To see if short-term price disequilibrium has existed in the past between Utah and other regional markets for fat cattle, we compared these shipment costs with actual price differences.

Results/Price Behavior

Price differentials between Utah and the three other regional markets appear to be highly seasonal (Figure 1). Prices in different markets fluctuate due to fluctuations in local supplies and demands for fat cattle (Shepherd and Futrell, 1982). These seasonal fluctuations can occur in competitive markets as long as the price differential does not exceed transportation costs. Sales of retail beef fluctuate from day-to-day and from week-to-week. The number of cattle available to be slaughtered in the local market also fluctuates on a daily basis. To keep his supply adjusted, a packer must raise or lower his bid price relative to other markets. When too many cattle are available, the packer lowers his price. When too few cattle are available the price rises. This may lead to wide fluctuations in price differences between markets from one day to the next or one week to the next. Packers also adjust bid prices due to changes in supply and demand for by-products, slaughter costs, and some type of profit target (Ward). Generally, Utah producers may expect relatively large price differentials in the late summer and early fall and relatively small price differentials in the late fall, early winter, and late spring (Figure 1). This indicates that the best possibilities for profitable shipment from Utah to other regions is probably during the late summer and early fall. No shipment decision should be made, however, unless actual price differences appear to be greater than shipment costs.

*This quote was for a load of 50,000 lbs. or approximately 45 head of 1,100 lbs. steers and/or heifers.

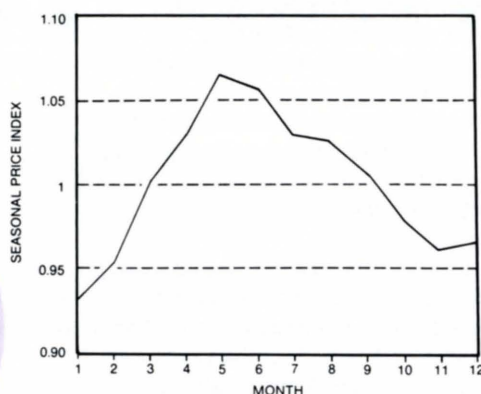


FIGURE 5. Seasonal variation in Omaha fat cattle prices, 1978-83.

Utah prices tend to experience much larger negative differentials than the other three regions. This may be due to several different factors, the first being transportation costs (both for live cattle and carcasses). The Utah market is relatively isolated, and consequently one would expect larger differentials from other regional markets for this reason. Also, there are few buyers of fat cattle in the Utah region. This perpetuates greater variations in Utah prices due to short-run surpluses or deficits of fat cattle for slaughter. This implies that the Utah market experiences short-run disequilibrium, and thus the local Utah differential may temporarily exceed actual transportation costs (plus shrinkage).

Tables 2 and 3 present the average prices and price differences for the four regions by year and month, respectively. Utah's price has been steadily declining relative to the other three regions during the past five years. The price difference between Utah and Texas is greatest in November and least in September. The difference between Utah and Colorado prices is greatest in November and least in January, while the price difference between Utah and Omaha is greatest in August and least in May. Prices in all four regional markets tend to peak in May and bottom in January (Figures 2-5). This seasonal pattern indicates greater demand and/or smaller supplies during the late spring and early summer and smaller demand and/or greater supplies of fat cattle during the late fall and winter months. Prices rise slightly in December, probably due to increased holiday demand. Prices in Utah tend to go slightly farther below average in October and

November relative to the other three regions. Prices in all four regions follow virtually the same seasonal pattern, indicating that seasonal pressures impact on all four regions with uniformity.

Shipment Costs Versus Price Differences

The weekly price differences between Utah and the other three regional markets were compared with estimated shipment costs (transportation and shrinkage costs). The number of times the price difference between Utah and the other three regions exceeded these shipment costs were determined using both full shrink and half shrink (Table 4).

As expected, the greater the distance between markets, the less probability there is that price differences will offset shipment costs. Based on this analysis, the Utah and Texas markets experienced short-term disequilibrium (price difference exceeded shipment costs) from 0 percent to 4 percent of the time during the past 5 years. Short term disequilibrium between Utah and Omaha existed from 0 percent to 2 percent of the time during the study period, while the Utah and Colorado markets were in disequilibrium from 4 percent to 14 percent of the time. The Texas and Colorado markets have shown an increased possibility for interregional trade from Utah during the past three years. The Colorado market shows the greatest probability for profitable trades from the Utah market. This would be expected since the Colorado market is the closest to Utah of the three other markets.

The increases in price differences between Utah and the other regional

TABLE 1. Estimated trucking costs for cattle in dollars per hundredweight.

From:	To:		
Logan, Utah	Denver, Colorado	Amarillo, Texas	Omaha, Nebraska
Year			
1978	\$0.89	\$1.24	\$1.35
1979	\$1.02	\$1.42	\$1.55
1980	\$1.20	\$1.67	\$1.83
1981	\$1.39	\$1.94	\$2.13
1982	\$1.49	\$2.08	\$2.28
1983 ^a	\$1.50	\$2.10	\$2.30

^aAs of July 23, 1983

markets for fat cattle (Tables 2 and 3), together with the disequilibrium between markets that occasionally occurs should be of interest to producers. One possible explanation of this phenomenon is that meat packers in the Utah area are able to exert some market power in their pricing activities, as judged by Utah's lower relative price. This cannot be considered conclusive, however, since other factors such as increasing transportation and slaughtering costs together with imperfect information may contribute to the present situation.

These results indicate that the Utah fat-cattle market is generally in equilibrium. Opportunities for interregional trade do occasionally exist, however, especially from the Utah market to Colorado. Increasing the amount and quality of information available to fat cattle producers should assist them in bargaining and also in transportation decisions. The main obstacle to better Utah prices appears to be the relative isolation and concentration of the market. Better marketing practices and information may help to offset part of this disadvantage.

Conclusions

The price relationships between Utah and three other regional markets for fat cattle were analyzed. Prices and price differences by month and year were charted and analyzed. The seasonal pattern of each of the markets was similar to the others, with lower prices for fat cattle in the late fall and winter and higher prices during the late spring and early summer (Figure 2-5). Price differences between Utah and the other three regions were found to be greatest in October and November and least in the late spring, early summer, and mid winter (Figure 1).

The meatpacking industry in Utah and Southern Idaho is highly concentrated.

TABLE 2. Mean prices and price differences for four regional markets for fat cattle by year, 1978-1983.^a

Year	UP	CP	OP	TP	UT	UC	UO
1978	52.33	52.79	52.77	52.56	-.22	-.45	-.44
1979	67.28	67.95	67.63	68.63	-1.35	-.67	-.35
1980	67.53	67.40	67.10	68.48	-.94	.13	.44
1981	64.07	65.01	64.24	65.96	-1.89	-.95	-.18
1982	63.56	65.31	65.14	65.93	-2.37	-1.75	-1.58
1983 ^b	63.58	65.63	64.88	66.06	-2.48	-2.05	-1.30

^aUP = Utah-Eastern Nevada-Southern Idaho price per cwt.
 CP = Colorado-Kansas price per cwt.
 OP = Omaha Price per cwt.
 TP = Texas Panhandle price per cwt.

UT = UP-TP
 UC = UP-CP
 UO = UP-OP
^bFrom January to July 1983.

TABLE 3. Mean prices and price differences for four regional markets for fat cattle by month 1978-1983.^a

Month	UP	CP	OP	TP	UT	UC	UO
January	59.48	59.81	59.18	60.69	-1.21	-.33	-.30
February	60.06	60.46	60.55	61.44	-1.38	-.39	-.48
March	62.91	63.44	63.49	64.02	-1.11	.53	.58
April	64.61	65.83	65.13	66.34	-1.74	-1.23	-.53
May	67.54	68.44	67.61	69.08	-1.54	-.859	-.07
June	67.07	67.89	67.11	68.33	-1.26	-.83	-.05
July	64.73	65.35	65.31	65.74	-1.01	-.62	-.58
August	63.30	64.34	65.14	64.63	-1.33	-1.04	-1.83
September	63.24	63.90	63.94	64.17	-.93	.67	.70
October	60.81	62.19	62.10	62.79	-1.98	-1.38	-1.29
November	60.24	61.96	61.04	62.77	-2.54	-1.73	-.81
December	61.72	62.38	61.28	63.19	-1.46	-.66	-.44

^aUP = Utah-Eastern Nevada-Southern Idaho price per cwt.
 CP = Colorado-Kansas price per cwt.
 OP = Omaha Price per cwt.
 TP = Texas Panhandle price per cwt.

UT = UP-TP
 UC = UP-CP
 UO = UP-OP

The level of concentration is high enough to foster suspicion that packers may be able to exert market power on producers. Utah prices are lower than other regional prices, and the differential has been increasing over time. Only for brief periods of time has this differential exceeded transportation costs. Our results indicate that any market power exerted by packers in Utah cannot exceed transportation costs. Even if price differentials are less than transportation costs, Utah prices follow the other prices closely, and disequilibrium will be quickly corrected.

This result speaks strongly for the existence of accurate and up-to-date information in the marketing of fat cattle and other agricultural commodities in Utah and other areas of the country. With Utah's relative isolation the effectiveness of information becomes increasingly critical. This is witnessed by the increased widening price differences between Utah and other areas. A producer who is aware of these price differences and the market possibilities around him should be able to profit from more effective marketing decisions through effective use of current market information.

TABLE 4. Total number of weeks price differences between Utah and other regional markets exceeded shipment costs between January 1978 and June 1983.

Region	Full Shrink	Excluding Excretory Shrink
Texas		
1978	0	2
1979	0	0
1980	0	0
1981	0	2
1982	0	6
1983 ^a	0	2
Omaha		
1978	1	4
1979	0	1
1980	0	0
1981	0	0
1982	0	2
1983	0	0
Colorado		
1978	5	7
1979	0	4
1980	0	0
1981	1	7
1982	3	14
1983	2	7

^aAs of July 23, 1983.

REFERENCES

- Andersen, Jay C., Roice H. Anderson, Rondo A. Christensen, Herbert H. Fullerton, Terrence F. Glover and Donald L. Snyder. "A study of meat packing in Utah," Utah Agricultural Experiment Station Research Report 72. September 1983.
- Miller, Steve. Miller's Transport, Inc. Hyrum, Utah. Personal Communication. July 28, 1983.
- Minish, Gary L. "Beef production and management." Reston Publishing Company. Reston, Virginia. 1982.
- Rutts, Jack. Statistician, Idaho State Department of Agriculture. Boise, Idaho. Personal Communication. October 1983.
- Shepherd, Geoffrey S. and Gene A. Futrell. "Marketing farm products." The Iowa State University Press. Ames, Iowa. 1982.
- Snyder, Donald L. "International cattle price dispersions: Information and competition." Economic Research Institute Study Paper #83-6. Utah State University, Logan, Utah. 1983.
- United States Dept. of Agriculture. "Livestock, Meat, and Wool Market News." Various Issues.
- United States Dept. of Agriculture, Economic Research Service. *Agricultural Outlook*. May 1983.
- United States Dept. of Commerce, Bureau of the Census. *Statistical Abstract of the United States 1982-83*. Washington, D.C. 1982.
- Utah State Department of Agriculture. "Fat cattle prices." Obtained from Don Carter. June 1983.
- Utah State Department of Agriculture. Utah Agricultural Statistics 1983.
- Ward, Clement E. "Slaughter-cattle pricing and procurement practices of meatpackers." U.S. Department of Agricultural Economics, Statistics and Cooperative Service. *Agricultural Information Bulletin* No. 432. December 1979.

ABOUT THE AUTHORS

Dee Von Bailey is an assistant professor in the Department of Economics at Utah State University. He is working on projects relating to pricing efficiency in cash and futures market.

B. Wade Brorsen is an assistant professor at Purdue University in the Department of Agricultural Economics. He is currently working on pricing efficiency in cash and futures market.

The Malnutrition

A close association appears to exist between diarrhea and malnutrition, although the actual roles various nutritional factors may play in diarrheal disease are just beginning to be determined. A major study of this important question has been underway through a cooperative effort of a research team composed of members of USU's Department of Animal, Dairy and Veterinary Science, Department of Biology, and Department of Nutrition and Food Science. The work was funded by the Utah Agricultural Experiment Station and by the Thrasher Research Fund of Salt Lake City, Utah.

Malnutrition continues to be a problem in many of the developing nations, and diarrheal disease is also widespread in such areas. Both conditions thrive in similar socioeconomic and cultural surroundings, and by acting together, contribute to high rates of childhood morbidity and mortality. Malnourished children have a higher incidence of severe diarrhea, resulting in high rates of diarrhea-related deaths (Mata et al., 1976; Scrimshaw et al., 1968; Wray, 1978). In addition to this human disease problem is the similar situation occurring in livestock throughout the world, wherein diarrheal disease, especially in the newborn, results in extreme economic losses. The role of nutrition in livestock diarrheal diseases is even less well understood.

The Rotavirus as a Cause of Diarrhea

While diarrhea in humans and livestock may be caused by a variety of agents, a particularly prominent culprit is the rotavirus. This virus (Figure 1) is a relatively small, wheel-shaped, ribonucleic acid-containing member of the Reoviridae virus family. It has been estimated by World Health Organization investigators that the rotavirus is associated with up to 50 percent of the

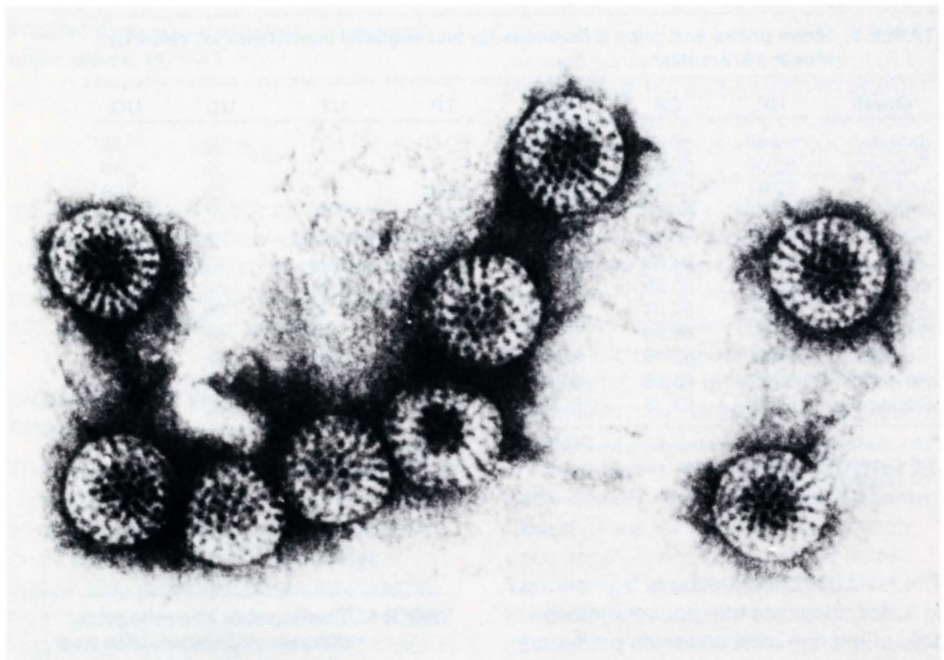


FIGURE 1. Electron micrograph of bovine rotavirus. Bar = 100 nm.

hospitalized cases of diarrheal disease (Rohde and Northrup, 1976, World Health Organization, 1978). In recent years the importance of the rotavirus has been similarly established in the etiology of diarrhea in young calves, piglets, lambs, foals, and other animals (Gillespie and Timoney, 1981).

Laboratory Model for Studying Rotaviral Diarrhea

To effectively study the rotavirus-induced diarrheal disease and the influence of nutritional factors on this disease, the USU researchers needed a laboratory animal model. The model had to mimic the disease seen in the human and livestock infants and be responsive to often major changes in diet. The animal that has proven ideal for such studies has been the laboratory mouse.

Experimentally infected infant mice, born to dams which had not had a prior exposure to the disease, develop a diarrheal disease which closely resembles the infection in both the human infant and in livestock. Within days after exposure to the rotavirus, the mouse exhibits a severe diarrhea and subsequently fails to gain weight at the same rate as its uninfected counterpart. Within one to two weeks after infection, this lessened weight gain becomes quite obvious and the infant appears runted (Figure 2). Occasionally the animals die from disease. Using a special enzyme-linked immunosorbent assay procedure, high virus titers can be measured in the intestines of the infant mice. The severity of the diarrhea can also be measured by determining the ratio of large intestine weight to whole mouse weight (Figure 3). Serum sodium levels

Diarrhea Connection

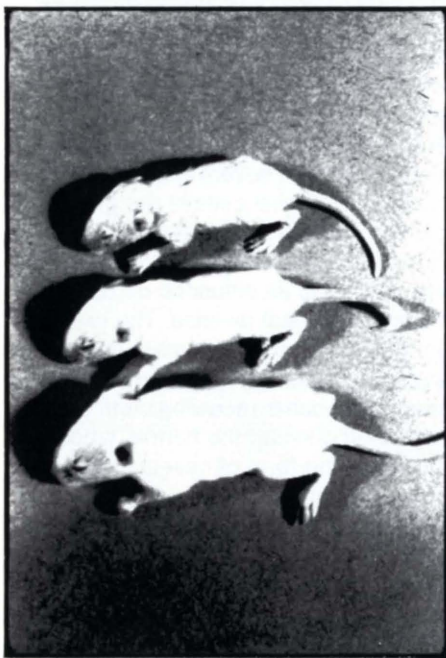


FIGURE 2. Weight loss and runting in mice caused by rotavirus infection. Upper two mice were infected 10 days earlier. The bottom mouse was uninfected.

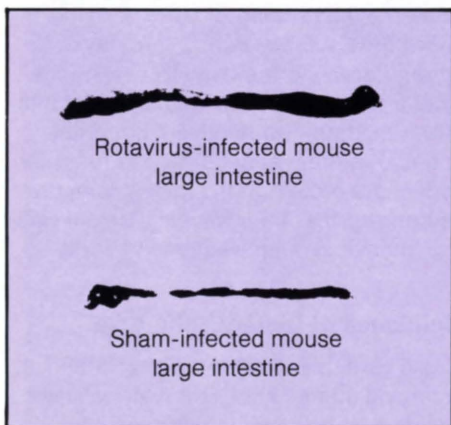


FIGURE 3. Comparison of intestines from rotavirus-infected and uninfected mice, illustrating the distention and enlarged appearance of the intestine caused by the infection.

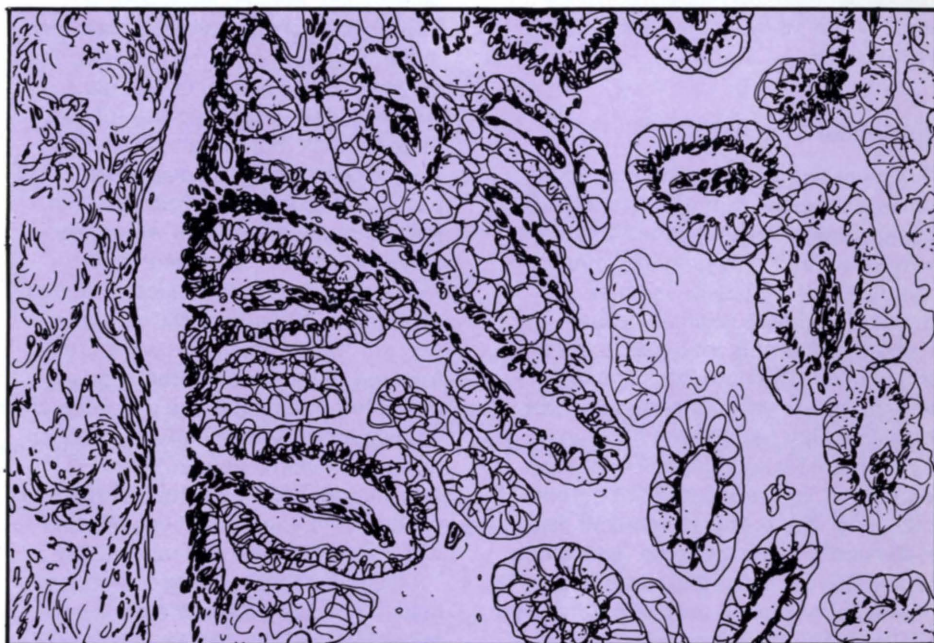
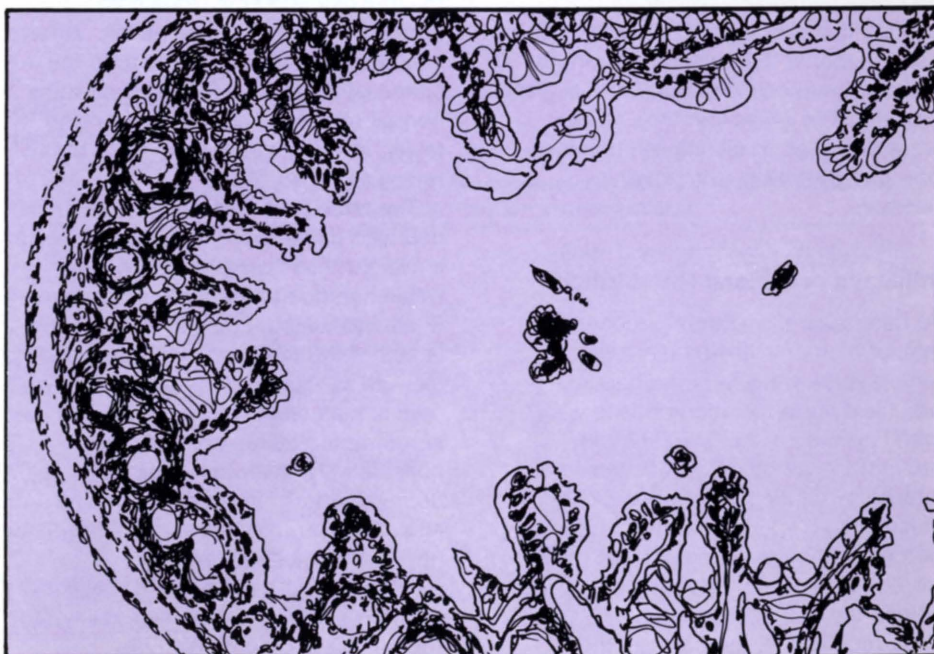


FIGURE 4. Destruction of intestinal villi caused by rotavirus infection. (a), normal, (b), infected. (Artists' rendition)



decrease in the infant mouse, indicating an electrolyte imbalance probably due to dehydration, an expected result of severe diarrhea. Histopathologic study of the intestines provided evidence of major destruction of the intestinal villi (Figure 4). Thus, assorted parameters are available in the mouse model for study of the diarrheal disease induced by rotavirus.

Nutritional Strategy

The initial strategy planned in the nutritional studies run with the rotavirus disease model in mice varied the target nutrient without changing the remaining components in the diet, using as a standard a dietary regimen recommended by the U.S. National Academy of Sciences. The nutrients selected were those that have affected specific immunological parameters such as humoral antibody titers, cell mediated responses, and resistance to bacterial infections. The nutrients selected also evidenced a tendency to be deficient in certain human diets, thus resembling situations that might exist in nature. As a follow-up to the initial studies, cross-fostering experiments, in which healthy mothers nursed infants from nutrient-deficient dams and the nutrient-deficient dams nursed healthy offspring from healthy mothers, were planned. These used nutrients that, when deficient in the diet, markedly enhanced the disease severity. The cross-fostering studies were designed to distinguish between pre- and post-natal nutritional influences.

Influence of General Malnutrition

To determine the effects of general malnutrition on rotaviral diarrhea, female mice were fed throughout pregnancy and lactation periods with a standard mouse diet in which the nutrient to calorie (N/C) ratio was altered by diluting the normal diet with glucose so it was 40 percent or 70 percent of normal. (Comparable mice were fed a normal, non-diluted diet for comparison purposes.) Thus the test mice received all the calories they

wished, but the nutritive value was lessened to a threshold point at which further reduction would have not been tolerated by the animals. Infants born to these dams were orally infected when 2 days old with rotavirus, and the extent of the subsequent infection was determined. Marked increases in severity of diarrheal disease was seen in the infants from dams receiving the 40 percent to 70 percent N/C diets (Table 1). This increased severity was evidenced as more deaths, reduced weight gain (Figure 5), and greater passage of diarrheic feces. Pre- and post-natal dietary effects were determined when the experiment was repeated, and newborn mice from dams receiving a 40 percent N/C or 100 percent N/C (normal) diet were cross-fostered to mothers fed the opposite diet. After infecting these cross-fostered infants, increased severity of infection was again seen (Table 2, Figure 6), although the effect was not as marked except when the 40 percent N/C diet was used both pre- and post-natally.

Gastrointestinal infection is normally held in check by the alimentary microflora and the cellular integrity of the gut. In malnutrition, slowed cellular turnover may cause mucosal thinning, altering the normal relationship between the mucosa and flora, thus compromising this biological barrier. When a viral infection causes damage to the epithelial lining, it must be repaired by normal mechanisms, which may be faulty, in the malnourished host, thus retarding repair.

The mice born to nutritionally deprived dams, were considerably lower in weight than normal and obviously weakened due to the malnourishment. Such weakening, coupled with the stress of the infection, no doubt caused them to succumb to the disease more readily than healthy baby mice. The serum antirotavirus antibody was considerably lower in nutritionally deprived dams, an expected finding since malnutrition has serious effects on host immunological defense mechanisms (Chandra, 1976, Faulk et al., 1976). Thus a decreased ability of the animal to defend itself im-

munologically may have also contributed to the enhanced severity of the diarrheal disease.

Influence of Dietary Protein

Protein deprivation has been known to enhance certain infections caused by parasites, bacteria, fungi, and other viruses (Chandra and Newberne, 1979) and, like general malnutrition, also strongly affects host immune response. Protein-undernourished subjects have also exhibited a severe depletion of mucosal epithelial cells in the gut. Thus protein was considered a likely nutritional factor to be next investigated with regard to its influence on rotavirus-induced diarrheal disease. The experiment was run in a similar manner to that done in the general malnourishment study, with dams receiving diets that contained one-half the normal amount of protein in the form of casein throughout pregnancy and lactation periods. Rotavirus-infected infants born to these dams again were more severely ill than those born to mothers receiving a normal amount of protein in their diet. This enhanced illness was especially apparent as a lessened weight gain (Figure 7), particularly later in the infection, and in a marked increase in deaths among the infants (Table 3). A significant increase in incidences of diarrhea was also evident.

An important observation in this study was the occurrence of dead or dying newborns, usually born 2 to 6 days prematurely, to the dams receiving the low protein diet. Such weakened infants from mothers fed relatively low quantities of protein accentuate the need for adequate protein in the diet. It is not surprising that the rotaviral disease was so intense in such weakened infants.

Influence of Dietary Folic Acid

Folic acid, also known as folacin or pteroylglutamic acid, is a water-soluble vitamin found particularly in green vegetables, beans, nuts and eggs. The vitamin is considered one of the first limiting nutrients in human diets (Leevy et al., 1965), particularly among

TABLE 1. Effect of pre- and post-natal general malnutrition on rotavirus infection in infant mice.^a

	Percentage Nutrients in Diet Relative to Control		
	40	70	100 (Control)
Number of mice	60	70	70
Average initial weight (g.)	1.8*	2.0	2.4
Mortality (%)	80**	48**	4
Average survival time of dying animals (days)	1.6	2.4	7.0

^aData from MS thesis by Roger L. Noble, Utah State University.
*P < 0.05 < **P < 0.001 (compared to control).

TABLE 3. Effect of pre- and post-natal dietary protein deprivation on rotavirus infection in infant mice.^a

	Percent Protein (Casein) in Diet	
	7.5	15 (Control)
Number of mice	36	73
Average initial weight (g)	1.3	1.8
Mortality (%)	76**	10
Average survival time of dying animals (days)	2.8	5.6
Incidence of diarrhea (%)	88**	50

^aData from MS thesis by Roger L. Noble, Utah State University.
**P < 0.001 (compared to control).

TABLE 5. Effect of pre- and post-natal dietary essential fatty acid deficiency and excess on rotavirus infection in infant mice.^a

	Fatty Acid Concentration (%)		
	25	100 (Control)	200
Number of mice	32	48	45
Average initial weight (g.)	3.1	3.6	3.4
Incidence of diarrhea (%)	100	100	100
Average intestinal virus titer	101	80	63
Intestine/whole body weight ratio, day 2 ($\times 10^{-2}$)	8.5*	7.2	7.6
Intestine/whole body weight ratio, day 8 ($\times 10^{-2}$)	9.8*	8.4	8.0
Average serum rotaviral anti- body titer (infants)	10*	40	38
Average serum rotaviral anti- body titer (dams)	26	64	36

^aData from MS thesis by Kamyar A. Zahedi, Utah State University.
*P < 0.05 (compared to control).

TABLE 2. Effect of cross-fostering to differentiate between pre- and post-natal malnutrition on rotavirus infection in infant mice.^a

	Percentage Nutrients in Diet Relative to Control			
	40 ^b /40 ^c	40/100	100/40	100/100
Number of mice	16	17	48	32
Average initial weight (g.)	1.7**	1.8**	2.0*	2.6
Mortality (%)	100**	57**	51**	0
Average survival time of dying animals (days)	1.4**	3.5**	4.7**	15

^aData from MS thesis by Roger L. Noble, Utah State University.
^bDiet of dams from which infants were taken.
^cDiet of fostering dams.
*P < 0.05 < **P < 0.001 (compared to 100/100 group).

TABLE 4. Effect of pre- and post-natal dietary folic acid deficiencies on rotavirus infection in infant mice.^a

	Folic Acid Concentration in Diet ($\mu\text{g/g}$)		
	0	0.125	0.5 (Control)
Number of mice	136	91	85
Average initial weight (g)	2.1	2.0	2.1
Folic acid content in uninfected mice ($\mu\text{g/g}$)	3.8*	5.1	5.3
Incidence of diarrhea (%)	73*	64	58
Average intestinal virus titer	544*	740*	386
Infants with no detectable rotaviral antibody (%)	81*	44	50
Day 20 weight gain (%)	410	450	440

^aData from PhD dissertation by John D. Morrey, Utah State University.
*P < 0.05 (compared to control).

TABLE 6. Effect of pre- and post-natal dietary zinc deficiency on rotavirus infection in infant mice.^a

	Zinc Concentration in Diet ($\mu\text{g/g}$)		
	4	12	60 (Control)
Number of mice	82	97	99
Average initial weight (g)	2.7	3.1	3.1
Zinc content in uninfected mice ($\mu\text{g/g}$)	15.2*	19.9	20.9
Thymus weight in uninfected mice (mg)	80.1*	92.3	99.8
Incidence of diarrhea (%)	71	65	75
Average intestinal virus titer	3596	5612	6618
Infants with no detectable rotaviral antibody (%)	20	0	4

^aData from PhD dissertation by John D. Morrey, Utah State University.
*P < 0.05 (compared to control).

pregnant women (Baker et al., 1975), and deficiencies in diets have had profound effects on both cellular and humoral immunity (Gross and Newberne, 1980). Folic acid was therefore considered as a dietary factor likely to influence rotavirus-induced diarrheal disease.

The experimental design of this study was similar to those used in the general malnutrition and dietary protein studies, with female mice fed folic acid-altered diets throughout pregnancy and lactation periods. In this study, the diets used included a diet containing essentially no folic acid, a diet containing 25 percent of a normally recommended quantity of the vitamin, and a normal diet that contained a full measure of folic acid. Infants from the lowest folic acid diet-fed dams had significantly lower folate levels in their livers, indicating a deficiency was achieved. In these same infants, a moderate enhancement of rotaviral disease was seen as determined by increased incidences of diarrhea (Table 4) and an increased number of animals containing high rotavirus concentrations in their intestines (Table 4). The infants from dams receiving the moderately reduced folic acid levels in their diet also were more severely ill than the mice from the dams fed the normal diet, although the intensity of the disease was not as great as among the lowest folic acid diet group (Table 4).

A general weakening effect was again seen in the infants from the group receiving the least folic acid. This weakening was especially apparent as significantly reduced weight gain among these infants (Table 4). Of particular interest was the significant number of infected infants from the lowest folic acid diet group who had no detectable serum antibodies to the rotavirus; this lack of antibody, a probable result of the lack of folic acid in the diet, may have been a major reason for the enhanced infection seen in these animals.

The diarrheal disease induced by rotavirus in the infant mouse appears to be enhanced by malnutrition as well as by deficiencies of specific nutritional factors.

Influence of Dietary Essential Fatty Acids

The essential fatty acids of particular interest in this research were linoleic and linolenic acids, both being polyunsaturated fatty acids found in corn oil. These fatty acids, like the other nutrients already discussed, affect antibody titers and cell-mediated immunity, depending on their level in the diet (Vitale and Broitman, 1981). They are also known to fluctuate widely in diets, so both lower than normal (25 percent) and greater than normal (200 percent) levels of these polyunsaturated fatty acids in the mouse diet were investigated with respect to their influence on rotaviral diarrhea. The diets were prepared using tocopherol-stripped corn oil as the source of the polyunsaturated fatty acids. Hydrogenated coconut oil, which has essentially no linoleic or linolenic acid, was mixed with the control diets. The infants from mice receiving the lower fatty acid diet had a more severe gastroenteritis than those mice receiving the normal diet. This enhanced severity was seen particularly as increased evidence of diarrhea (intestine/whole body weight ratios, Table 5) and more marked histopathological changes in the intestinal epithelium. These animals also exhibited lower serum antibody titers to rotavirus than

did mice in the normal diet group. Interestingly, the animals from the 200 percent fatty acid diet group, while not visibly exhibiting signs of increased diarrheal disease, had more extensive damage to the intestinal mucosa as measured by the histopathology of stained intestinal sections.

Influence of Dietary Zinc

Deficiencies of dietary zinc have been associated with impaired immune responses in numerous studies, and dietary zinc deficiency occurs quite frequently in many countries (Fraker and Leucke, 1981). The influence of dietary deficiencies of this mineral on the rotavirus disease was therefore determined using the same procedure as described above. Effects of a highly deficient diet, containing 4 μ g zinc per gram, and a moderately deficient diet, containing 12 μ g of zinc per gram, were compared with those of a "normal" diet containing 60 μ g of zinc per gram. The deficient diets caused an obvious zinc deficiency in the infant mice (Table 6), as evidenced by reduced thymus weights, a lesser ability of the animals to gain weight, a lower measurable zinc concentration in the whole animal, and the presence of scaly skin. Despite these general manifestations of zinc deficiency, however, the animals displayed no apparent increase in severity of the diarrhea disease using any of the parameters described earlier. Antibodies to the rotavirus were at approximately the same levels in all three groups of animals. The reason for this apparent and surprising lack of effect is still under investigation.

Conclusions

The mouse has proven ideal for investigation of rotavirus-induced diarrheal disease. The disease elicited in the infant mouse closely resembles the gastroenteritis seen both in human

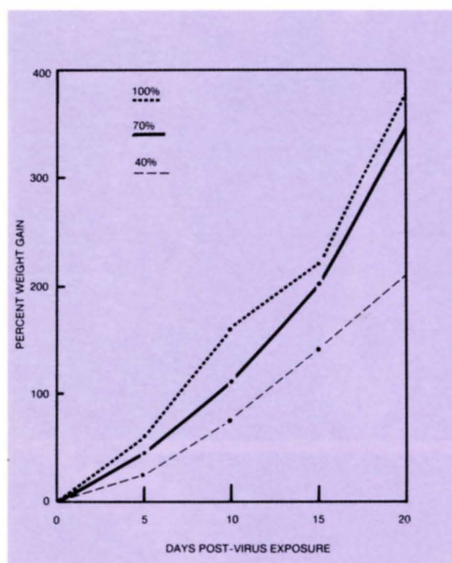


FIGURE 5. Weight gain of rotavirus-infected infant mice from dams receiving 40, 70, or 100% nutrient/calorie diets.

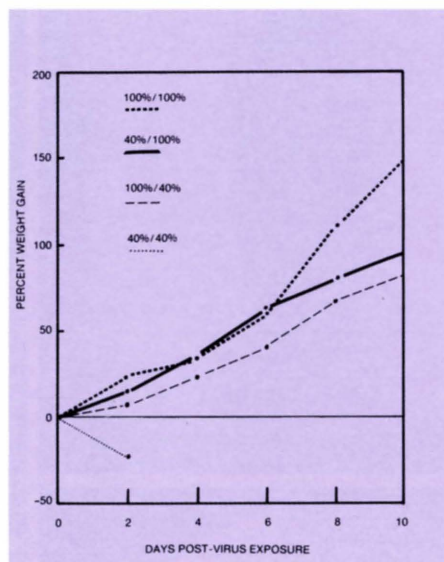


FIGURE 6. Weight gain of rotavirus-infected infant mice cross-fostered from 100% N/C-fed dams to other N/C dams (100%/100%), from 40% N/C dams to 100% N/C dams (40%/100%), from 100% N/C dams to 40% N/C dams (100%/40%), and from 40% N/C dams to other 40% N/C dams (40%/40%).

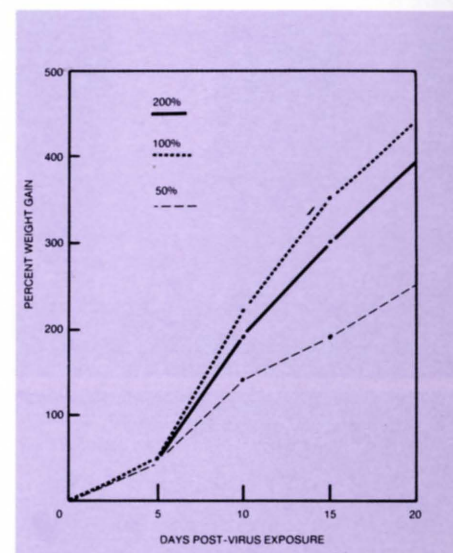


FIGURE 7. Weight gain of rotavirus-infected infant mice from dams receiving low (50%), normal (100%) or high (200%) protein (casein)-containing diets.

infants and in newborn livestock, with many easily measurable parameters available to the investigator.

The diarrheal disease induced by rotavirus in the infant mouse appears to be profoundly enhanced by malnutrition as well as by deficiencies of specific nutritional factors such as protein, folic acid, and polyunsaturated fatty acids, which may also be found lacking in human and possibly livestock diets. Our knowledge of the mechanisms by which this disease enhancement occurs is incomplete, but the increased disease severity appears to be primarily involved with a general weakening of the newborn infant as well as with inhibition of key immunological mechanisms the infant needs to resist the infection.

Such data strongly point to the need for the prospective mother to be provided with a well-balanced diet throughout her pregnancy if her offspring are to adequately resist the ravages of the widely occurring rotavirus-induced diarrheal disease.

REFERENCES

- Baker, H., Frank, O., Thomson, A. D., Langer, A., Munves, E. D., De Angeles, B. and Kaminetzky, H. A. 1975. Vitamin profile of 174 mothers and newborns at parturition. *Am. J. Clin. Nutr.* 28:59-65.
- Chandra, R. K. 1976. Reduced secretory antibody response to live attenuated measles and poliovirus vaccines in malnourished children. *Brit. Med. J.* 2:583-585.
- Chandra, R. K. and Newberne, P. M. 1979. *Nutrition, Immunity and Infection: Mechanisms of Interactions*, pp. 1-246, Plenum, New York.
- Faulk, W. P., Pinto-Paes, R. and Marigo, C. 1976. The immunological system in health and malnutrition. *Proc. Nutr. Soc.* 35:253-260.
- Fraker, P. J. and Leucke, R. W. 1981. Effect of dietary zinc deficiency on lymphocyte function in the mouse. pp. 107-120. In: *Diet and Resistance to Disease*. Plenum, New York.
- Gillespie, H. J. and Timoney, J. F. 1981. *Hagan and Bruner's Infectious Diseases of Domestic Animals*, pp. 644-648. Cornell Univ. Press, Ithaca.
- Gross, R. L. and Newberne, P. M. 1980. Role of nutrition in immunologic function. *Physiol. Rev.* 60:188-302.
- Leevy, C. M., Cardi, L., Frank, O., Gellene, R. and Baker, H. 1965. Incidence and significance of hypervitaminemia in a randomly selected municipal hospital population. *Am. J. Clin. Nutr.* 17:259-271.
- Mata, L. J., Kronmal, R. A., Garcia, B., Butler, W., Urrutia, J. J. and Murillo S. 1976. Breast-feeding, weaning and diarrhoeal syndrome in a Guatemalan Indian village. p. 311-338. In: *Acute Diarrhea in Childhood*. CIBA Foundation Symposium 42, Elsevier, Amsterdam.
- Rohde, J. E. and Northrup, R. S. 1976. Taking science where the diarrhoea is. pp. 339-358. In: *Acute Diarrhea in Childhood*. CIBA Foundation Symposium 42, Elsevier, Amsterdam.
- Scrimshaw, N. S., Taylor, C. E. and Gordon, J. E. 1968. Interactions of nutrition and infection. *WHO Monogr. Series* 57, pp. 1-85.

Vitale, J. J. and Boritman, S. A. 1981. Lipids and immune-function. *Cancer Res.* 41:3706-3710.

World Health Organization. 1978. Control of diarrheal diseases: WHO's programme takes shape. *WHO Chron.* 32:369-372.

Wray, J. D. 1978. Direct nutrition intervention and the control of diarrheal diseases in pre-school children. *Amer. J. Clin. Nutr.* 31:2073-2082.

ABOUT THE AUTHORS

Robert W. Sidwell is a Research Professor with joint appointments in the Department of Animal, Dairy and Veterinary Sciences and in the Department of Biology. Prior to coming to Utah State University, he was Director of the Nucleic Acid Research Institute in Irvine, California. His professional interest is in the control of virus diseases.

Bill B. Barnett received a PhD in biochemistry from Utah State University, and is presently Research Associate Professor in the Department of Biology. His main areas of research include the study of rotaviruses and clinical procedures for virus detection.

Arthur W. Mahoney received his PhD degree from the University of Maine in 1965 and has worked on appetite regulation at Harvard University. He is Professor of Nutrition and Food Sciences with research interests especially in food iron bioavailability.

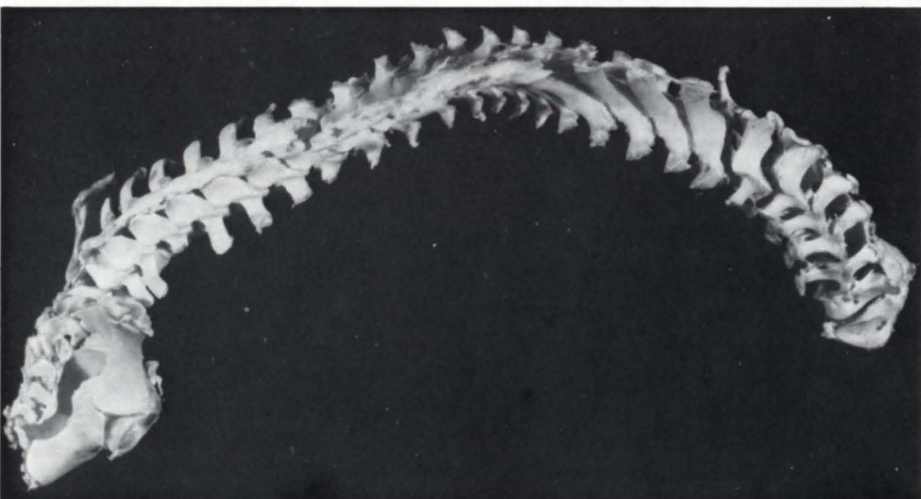
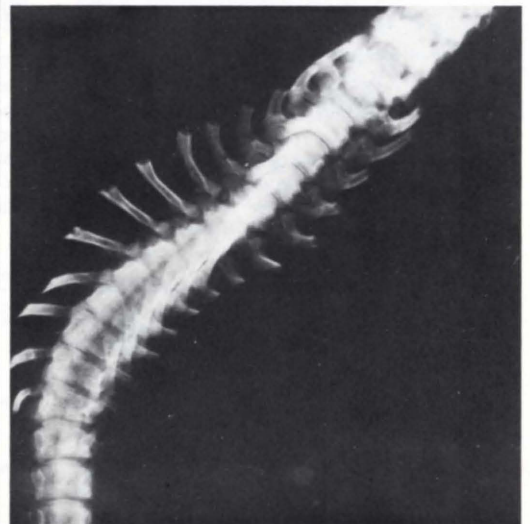
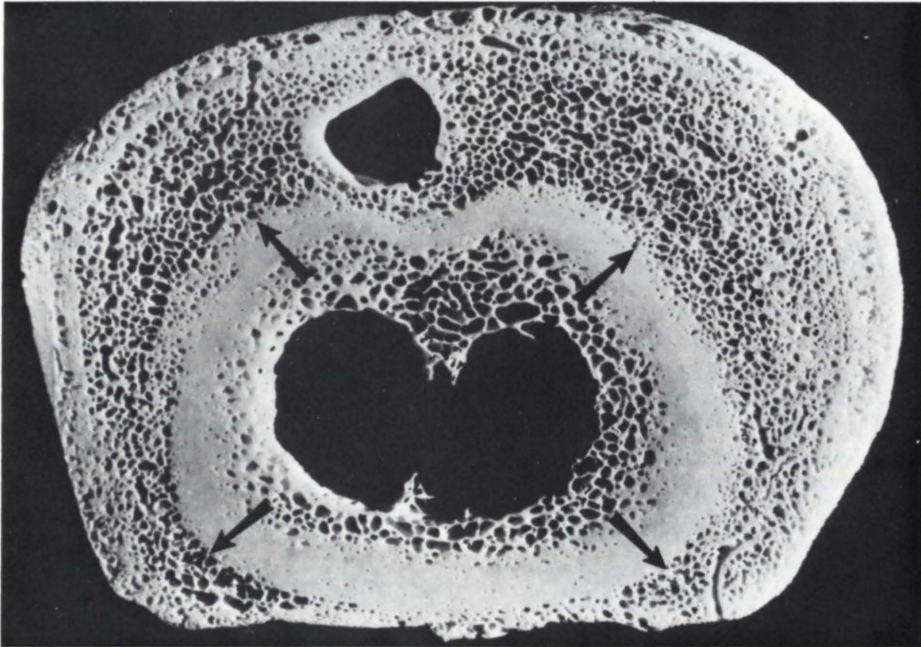


FIGURE 1. Bovine metatarsal bones. One on the left is normal. The other three show increasing gradations of fluoride induced bone effects.

FIGURE 2. Cross section of bone on the right is Figure 1. Arrows indicate surface of original bone. There is a loss of bone on the inside and excessive proliferation of bone on the outside.

FIGURE 3. Humerus, radius, and ulna bones from a young hereford calf. The bones on the left are normal. Those on the right are deformed. The calf's dam ingested lupine plants while pregnant.

FIGURE 4. Radiograph of spinal column of a calf showing abnormal curvature and rotation. The calf's dam ingested lupine plants while pregnant.

FIGURE 5. Spinal column from calf with inherited defects similar to those induced by lupine.

J. L. SHUPE, A. E. OLSON, N. C. LEONE, E. J. GARDNER and Y. ELSNER

COMPARABLE BONE DEFECTS IN ANIMALS AND HUMANS

Many forms of congenital (present at birth) defects, ranging from minor changes to striking abnormalities in the newborn have been observed. The cause and pathogenesis (development) of congenital anomalies are complex.

The musculo-skeletal system is structurally and functionally a unit. It can be completely understood only if this concept is kept in mind while artificially separated parts are studied. This unity is reflected in the close inter-relationship between pathologic changes of bone, joints, and muscles. Whenever one of the three major components of the locomotor system becomes abnormal, the other two ultimately undergo secondary changes. The mechanical continuity of the main constituents of the musculo-skeletal system and their smooth function are made possible by the minor constituents of this system and their remarkable adaptation to local needs. These other, less prominent structures include the tendons, tendon sheaths, and bursae.

Bone is a hard, highly specialized form of connective tissue composed of branching cells and intercellular substance. The hardness results from deposition of a complex mineral substance (chiefly composed of calcium, phosphates and carbonates) within a soft organic matrix or form. To understand diseases of bones one must know and understand bone structure and function. The major functions of bone are mechanical (support, locomotion, and protection), metabolic (homeostasis storehouse for minerals and fat), and as a site for production of blood cells.

Bone structure and composition varies depending upon age, function, metabolic state, use, state of health, and other factors. The shaping and size of bones (modeling) stops at maturity. Remodeling, or the removal and replacement (turnover) of bone goes on throughout life. The major and number-one authority on bone is "bone" and it "behooves us" to properly observe and interpret what is happening. In the evaluation and understanding of diseases of bones one must appreciate and realize that various phases of bone activity vary and may not and usually do not represent the steady state of bone activity. One must beware of applying cellular level responses to interpret tissue, organ, system, and total-body responses.

Remarkable recent revival of interest in the problems of bone on the part of investigators in all fields of basic science and clinical medicine has occurred. The study of bone dynamics and skeletal kinetics is

extensive beyond the possibility of complete understanding by one person.

Abnormalities of the musculo-skeletal system are numerous. The magnitude of the defects vary widely. The changes may be diffuse, localized, or appear as single isolated anomalies. Only musculo-skeletal structures may be affected, or other tissues, organs, or systems may also be involved.

Musculo-skeletal defects may occur for a variety of reasons but the causes are frequently divided into genetic and environmental. Inheritance may be via many genetic modes or patterns. Environmentally, abnormalities may be induced nutritionally by many different elements or compounds. These elements or compounds may appear in excess or as deficiencies. The compounds may either exist naturally in feedstuffs or be manufactured and introduced into the food chain. The compounds responsible are often ingested by pregnant animals and affect the unborn offspring during embryonic development and fetal growth. Musculo-skeletal defects may also be acquired by an animal through intrauterine infections, maternal endocrine imbalances, trauma, or physical phenomena.

Cattle have the highest incidence of reported skeletal malformations in livestock. Historically, one of the first widely recognized musculo-skeletal defects in cattle was dwarfism which affects the entire animal. Dwarfism has been associated with certain breeds.

Several musculo-skeletal defects of livestock have been studied at Utah State University (USU).

In the early 1950s, USU researchers responded to requests from farmers, ranchers, civic officials and industrial representatives to diagnose a chronic insidious condition affecting the teeth and skeletal system of numerous domestic animals and some of the wild animals in Utah and Salt Lake counties. A definitive diagnosis of fluoride toxicosis (fluorosis) was confirmed after extensive and detailed clinical examinations of affected animals were conducted. Long term controlled studies were initiated and demonstrated that developing teeth and bones of animals of all ages were particularly susceptible to effects of excessive intake levels of fluoride. It was also proven that fluorides had no congenital malformation or tumor-inducing effects. Factors that affect expression of the different types and various degrees of bone changes (osteofluorosis), as illustrated in Figures 1 and 2 were determined. Information developed

at USU has been used to establish recommended standards and criteria for fluoride and is being utilized worldwide to aid in the diagnosis, evaluation, and prevention of fluoride toxicosis in animals. Some of the results of USU animal studies have been used by researchers to reinforce their recommendations of the beneficial effects of ingestion of proper amounts of fluorine by humans.

During the 1960s a deformity of Hereford calves that included persistent flexure or contracture of limbs (arthrogryposis), dorsal curvature with a humpback appearance (kyphosis), lateral deviation of the spinal column (scoliosis), twisting of the neck (torticollis) and cleft palate was studied. At the same time there was much research effort being directed to studying similar effects caused by ingestion of certain vegetation by pregnant cows (Figures 3 and 4). Repeated matings of a certain bull with a small group of cows over several years led to the conclusion of an inherited musculo-skeletal defect very similar in appearance to the deformities induced by ingestion of lupine and some other plants (Figure 5).

An appeal for help from a prominent Jersey breeder led to the identification of a condition popularly called "Limberleg." The cause proved to be a hereditary recessive factor and only resulted when inbreeding of certain family lines was practiced. The USU studies led to many subsequent reports to the breed association of the birth of Jersey calves fitting the limber leg description.

There have been other studies conducted at USU on musculo-skeletal defects. A factor influencing the study of musculo-skeletal abnormalities is the presence on the USU campus of the Western Regional USDA Poisonous Plant Laboratory. Ingestion of some native range plants by pregnant animals may result in congenital abnormalities of the musculo-skeletal and other systems. Causes of these disorders must be differentiated from heritable conditions and inherited conditions must be differentiated from environmentally induced defects.

Currently, studies are continuing at USU on a long-term comparative study of Hereditary Multiple Exostoses (HME). These multiple bone tumors are usually benign. No sarcomatous (malignant) transformations of these tumors have been detected to date in any of our studies with either the horses or humans (Figures 6 and 7). Other researchers have reported occasional malignancies among their human study groups.

HME is a skeletal disorder that primarily affects endochondral bones. Endochon-

dral bones are bones that form from cartilaginous models. A similar condition has also been identified in dogs, cats, lizards, and lions. Some of these incidents were reported in the literature as simple case histories of an examination of a single individual and others were followed to confirm an inheritance pattern.

The inheritance pattern of a single dominant autosomal gene (non sex linked) has been observed in all pedigrees studied at USU for HME in both horses and humans. Affected individuals transmit this trait to approximately 50 percent of their progeny, in keeping with the mendelian segregation of alleles, whereas nonaffected individuals do not transmit the condition to their offspring.

The tumors in affected horses are most often present at birth and in affected humans are usually identified after 1 year of age. Skeletal development at birth probably accounts for this timing variation. In both horses and humans, the tumors vary in size, shape, and texture (Figures 8 and 9). The lesions tend to be somewhat bilateral. The skull is not involved in either species. The tumors become more obvious during periods of active skeletal maturation and cease to enlarge after maturity (closure of the growth plates). They are usually found in areas where bone growth is most active and areas subjected to greatest stress and strain. The exostoses are usually disfiguring and may interfere with some types of physical performance, infringe on some blood vessels, and induce pain by exerting pressure upon surrounding structures such as tendons, nerves, the spinal cord, and some visceral organs (liver and kidney). HME is characterized on gross examination as a deformity or tumor of bone with a cartilage-cap. Pathologists call this type of tumor an osteochondroma. Radiographically, the observed tumors had varying degrees of mineralization, were distinct or occasionally appeared as poorly delineated lesions. Histopathologic features varied somewhat depending on age, location, and size of the tumors but generally were those of an osteochondroma in both species. Periosteal and other connective tissues usually covered cartilage-capped spongy chondro-osseous (mixed cartilage and bone) growths (Figure 10). Subchondral foci of cartilage cells were located within a haphazardly arranged and poorly organized trabecular network.

Sarcomatous transformations have not yet been detected after 15 years of USU study in horses, although such changes have occasionally been reported in studies by other authors of HME in humans. Estimates of the incidence of sarcoma-

tous transformation in humans vary from 1 percent to 20 percent. Factors such as variability in criteria used for differential or definitive diagnosis of HME, age group, and mode of study have had important bearing on the percentage of reported sarcomatous transformations.

No difference occurred in the severity of clinical manifestations of HME between males and females. Type and distribution of lesions showed no difference in sex or family lines. No trend was observed for changes in the severity or multiplicity of the lesions in successive generations. Some human families in the USU study include up to 5 generations.

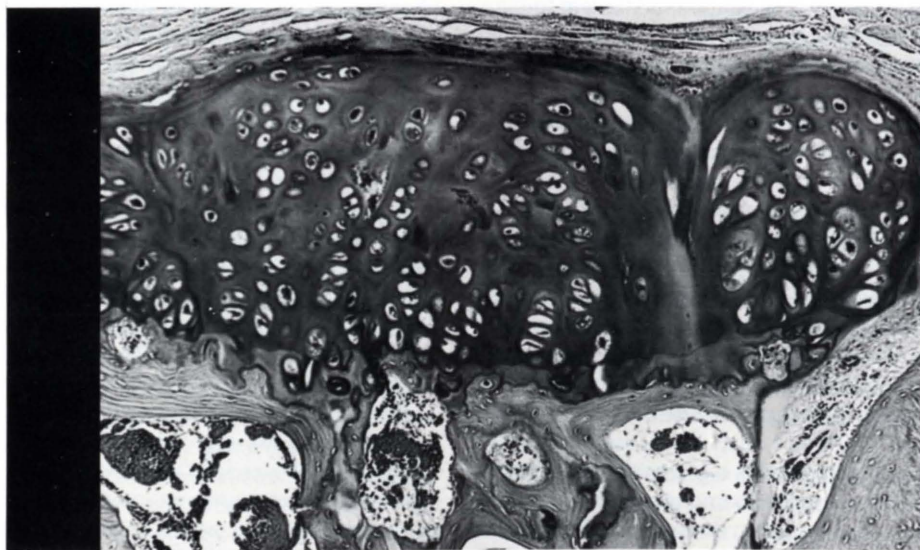
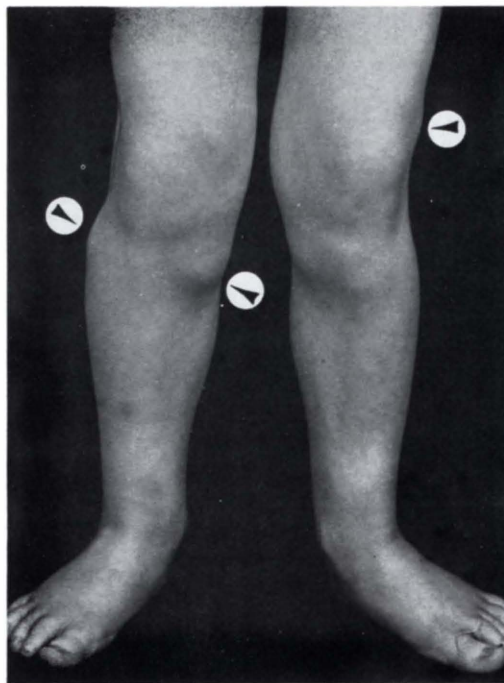
The clinical, gross, and microscopic appearance of the lesions in horses simulates that of the lesions of HME in humans. Thus, comparative studies of horses and humans, with follow-up evaluations, can provide information on the cause and development of the disease, including the incidence of malignancy. To achieve this end, researchers at USU have been studying affected members of both species.

Fourteen human family groups evidencing HME are under investigation. One family group of 48 contains 22 HME affected individuals. The pattern of single gene, autosomal dominant inheritance and the phenotypic expression (appearance) are similar to those in the horses that are also under investigation.

There is still much research activity on HME at USU. Horse and human patients in the study are periodically re-examined to study tumor development and check for possible sarcomatous transformation. Individuals born in the study groups are evaluated for tumor appearance, growth and development.

Newly developed molecular biological techniques are being utilized to identify a particular gene in the DNA. Purified DNA is being isolated from HME affected horses and humans. Enzymes are used to fragment the DNA and possible polymorphisms (many form) are located. Studies are beginning to utilize isotope labeled markers for detection of genes.

Additional valuable information is being obtained on tumor metabolism by feeding Sodium Fluoride to HME affected horses. Dosages of up to 7.5 mg of Fluorine/kg of body weight/day have been fed to some of the horses for as long as eight years. Uptake of fluorine by the tumors is about 2 times that of normal bone in the same animals but no adverse morphological (structural) effects have been noted to date. This phase of the study is closely integrated with observations for possible sarcomatous transformations.



Since the symptoms as well as the pathologic aspects and genetics of HME in horses and human beings are similar, results of experiments on horses have had valuable applications to humans.

FIGURE 6. Three-day-old colt with tumors (HME) as indicated by arrows.

FIGURE 7. Lower legs of a 5-year-old boy with tumors (HME). Sites of tumors are indicated by arrows.

FIGURE 8. Lower end of radius from a 3-year-old horse with prominent tumors (arrows) of HME.

FIGURE 9. Rear view of knee area of a man 67 years of age with HME. Most prominent tumors are indicated by arrows.

FIGURE 10. Photomicrograph of a small tumor from a horse with HME. At top, abnormal cartilage cells (dark area) overlay abnormal bone below. (H & E stain, $\times 75$)

REFERENCES

- Gardner, E. J., Shupe, J. L., Leone, N. C., Olson, A. E.: Hereditary multiple exostosis: Comparative genetic evaluation in man and horses *J. Hered.* 1975, 66:318-322.
- Shupe, J. L.: Hereditary multiple exostoses, *Equine Medicine and Surgery*, 2nd edition. Edited by E. J. Catcott, J. R. Smithcors. Wheaton, Illinois, American Veterinary Publishers, 1972, pp. 501-505.
- Shupe, J. L., Leone, N. C., Gardner, E. J., Olson, A. E., Johnson, L. C., Gambardella, P. C.: Hereditary multiple exostosis Spontaneous Animal Model of Human Disease. Vol II. Edited by E. J. Andrews, B. C. Ward, N. H. Altman, New York, Academic Press, 1979, pp. 223-228.
- Shupe, J. L., Leone, N. C., Olson, A. E., Gardner, E. J.: Hereditary multiple exostoses: Clinicopathologic features of a comparative study in horses and man. *Am. J. Vet. Res.* 1979, 40:752-757.
- Jaffe, H. L.: Tumors and Tumorous Conditions of Bones and Joints. Philadelphia, Lea and Febiger, 1958, pp. 150-167.
- Virchow, R.: Pathologie des Tumeurs (Trans. German to French). Germer Bailliere, Librairie edition. Paris, P. Aronssohn, 4 vol), 1867-1871.
- Aegerter, E., Kirkpatrick, J. A.: Orthopedic Diseases, 3rd edition. Philadelphia, W. B. Saunders 1968, pp. 122-144.

ABOUT THE AUTHORS

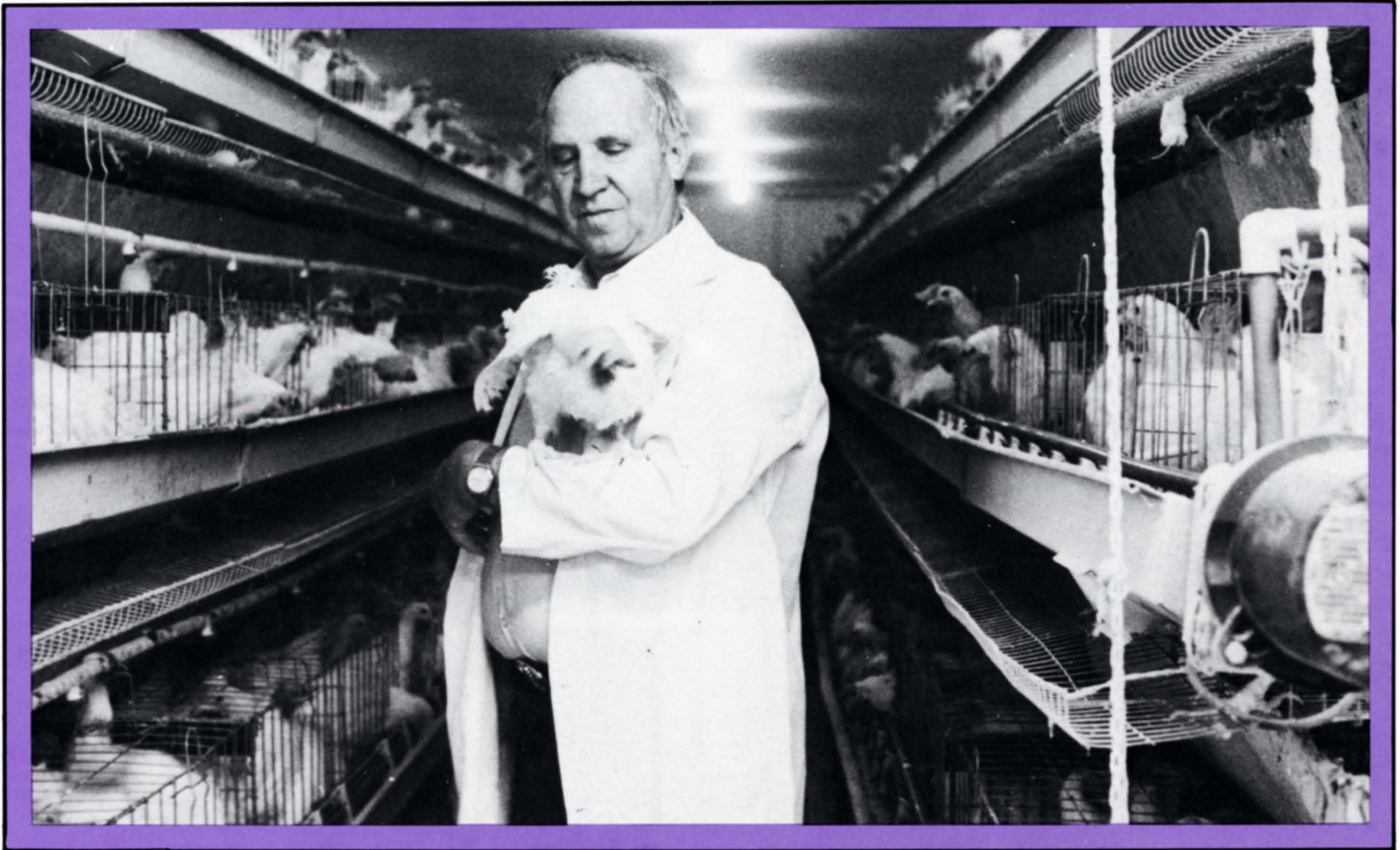
James L. Shupe is a professor of pathology and toxicology in the Department of Animal, Dairy and Veterinary Science.

Arland E. Olson is a research associate of histotechnology in the Department of Animal, Dairy and Veterinary Science.

Nicholas C. Leone, M.D., is an adjunct professor of comparative medicine in the ADVS Department at USU. He was formerly the Chief of Medical Investigation in NIDR, National Institute of Health. He resides at Fort Lauderdale, Florida.

Eldon J. Gardner is professor emeritus, Department of Biology at USU.

Yoko Y. Elsner is a research supervisor in the ADVS Department at USU.



A FEED SOURCE OF THE FUTURE

Animal wastes represent a potential major recoverable feed resource which at present is being under-utilized. Assuming animal wastes contain 15 percent dry matter, the annual dry matter yield in the U.S. would approximate 330 million tons (Fontenot and Jurubescu). There would be about 7 million tons per year produced from poultry alone.

Utah could produce about 56,000 tons per year from our combined turkey and egg production units. Not all of this is readily available, however, without some major changes in rearing methods, especially by the turkey industry. The egg industry, on the other hand, could have available about 28,000 tons per year with development of an economical method of reducing the water content of cage layer feces.

Program Not New But Intensified

The feeding of poultry wastes to animals is not new. The program has simply been

intensified in the last 15 to 20 years. Broiler litter has probably received the most emphasis because of the ease of collection and processing. Anthony has stated that, "Beef cattle producers searching for ways to lower feed costs and/or stretch feed supplies should consider broiler litter as a possible nutrient source for wintering, growing, and finishing rations, broiler litter is a valuable source of crude protein and minerals for beef cattle." Cattle producers in Arkansas (Cope-land) have survived three very dry years by feeding broiler litter. One grower stated that it is the cheapest way to put weight on weaned calves and to winter a cowherd. In Utah, there is no broiler industry, but there is an egg and turkey industry that have waste products.

Cage layer waste is one of the more difficult products to dispose of, use, or process because of its high moisture content (70-80 percent). Some management problems have added to the problem. These have included watering system failures

that permitted more water to run into the droppings, thus causing the material to become corrosive and have offensive odors. Fresh poultry waste from a normal healthy flock of birds has a minimum of offensive odors. The highly offensive odors develop as the feces are permitted to undergo putrefaction. Except for broiler and turkey litter, poultry wastes are wet as collected and the presence of moisture is the greatest restriction on the use of wastes in animal feeding programs.

According to Anthony, representing the scientific community, animal wastes should be processed prior to use as animal feed. Animal waste has greater value as animal feed than it has for any other current method of utilizing the waste products. General acceptance of animal waste feeding has not occurred within the agricultural community.

Several methods of processing cage layer waste have been explored and it appears that no one way will be the best method. Each area will need to examine



Machine used to process layer waste. "Protein source" produced contains about half layer waste; the rest is salt, minerals, and barley.

its natural advantages and capitalize on them. It is also essential to determine where processed waste fits into the food cycle of animals.

Dehydration Methods

One method to process cage layer waste into products that can be used for animal feeding involves dehydration, using fossil fuels for heating. Egg producers who are using this process are finding the high cost of gas and oil to be a deterrent. The odors from fossil fuel dehydration can be strong and offensive to neighbors. Currently the commercial industry is looking for more economical and feasible methods of utilizing poultry wastes.

Ensilage

Another method of utilizing cage layer wastes has been to blend wastes with corn silage and other feed ingredients and ensile them together. Workers have shown with proper ensiling that the product is palatable to cattle, free of pathogenic disease organisms and aesthetically acceptable for most livestock producers (Anthony). Ensiling has certain limitations because there are chickens voiding excreta everyday and silage is harvested once a year. The ensiling process would not be possible in the colder climates during the winter. One operation in Mississippi appears to be successful in utilizing cage layer waste on a year-round basis. This operation re-ensiles corn silage blended with cage layer waste, molasses, vitamin A, and dry brooder house litter. The dry brooder house litter was added to reduce the moisture. This operation had 1,000 head of feeders receiving the re-ensiled product. They reported that the cattle did quite well and made economical gains.

TABLE 1. Nutritional value¹ of animal wastes.

Item	Kind of Waste			
	Broiler waste ²	Dehydrated caged layer waste ²	Composted caged layer waste ³	Fresh dried caged layer waste ³
Crude Protein %	31.3	28.	40.	40.
True Protein %	16.7	11.3	—	—
Digestible Protein %	23.3	14.4	—	—
Crude Fiber %	16.8	12.7	—	—
Ether Extract %	3.3	2.	—	—
NFE %	29.5	28.7	—	—
Dig. Energy (ruminants) kcal/g	2440.	1884.	—	—
Metab. Energy (ruminants) kcal/g	2181.	—	—	—
TDN (ruminants)	59.8	52.3	—	—
Ash %	15.0	28.	—	—
Calcium %	2.4	8.8	8.5-11.0	—
Phosphorus %	1.8	2.5	2.0- 2.3	2.0
Magnesium %	0.44	0.67	—	—
Sodium %	0.54	0.94	0.70	—
Potassium %	1.78	2.33	—	—
Iron, ppm	451.	2000.	1575.	—
Copper, ppm	98.	150.	85.	—
Manganese, ppm	225.	406.	317.	—
Zinc, ppm	235.	463.	290.	—

¹Dry matter basis.

²Adopted from Bhattacharya and Taylor (1975).

³Determined from fresh product (3 day collection by Soil, Plant and Water Analysis Laboratory, U.S.U.).

TABLE 2. Influence of storage¹ time of waste in laying house² on nitrogen content of feces.

Storage Time	Moisture Content	Nitrogen Content ³	Calcium Protein Content
	%	%	%
3 days	58.4	5.72	35.8
13 weeks	46.8	2.94	18.4

¹Storage was in the same commercial layer house under relatively "dry" conditions.

²The laying house was the deep pit style house.

³Nitrogen content was determined on dried samples.

TABLE 3. Results of feeding CLW to breeder crossbred heifers.

Treatment	Gain per day by periods ¹ Utah State University, 1981			
	1st	2nd	3rd	4th
	lbs.	lbs.	lbs.	lbs
Alfalfa control	1.17	0.50	1.25	0.57
Composted CLW	0.60	0.73	2.13	0.60
Mechanically composted CLW	0.90	1.03	2.60	0.66

¹30-day intervals.

Composting

Another method is that of composting in which volatile solids, such as those in animal manure, are digested by bacterial action. Composting may be done under anaerobic and/or aerobic conditions. The aerobic method is most commonly used to eliminate odors and reduce the volatile solids, as rapidly as possible, to a stable state.

Aerobic composting of animal wastes is improved by regulating the moisture at 45 to 50 percent. Biological activity increases as the temperature of the compost increases. Optimum temperature for composting range from 120 to 160°F. Under controlled moisture, temperature, and aeration conditions, composting time may be reduced.

Once the compost becomes stabilized, it is relatively odorless and may be stored without attracting flies or rodents. Also, most of the pathogens have been killed

during the composting process. The final compost has a volume of approximately one-fifth to one-fourth that of the initial waste.

Composting of cage layer waste has gained attention in Utah with two different methods. One being the "natural" method, the droppings are removed from a layer house and piled in a row about three feet deep and eight feet wide. This process has been developed by Howlett Egg Farms near Elberta, Utah. To duplicate their program there must be at least three conditions met:

1. Manure must be low in moisture; approximately 50-53 percent (normal 70 percent).
2. The farm must be located in an area that has extremely low rain fall.
3. The temperature must be relatively warm. Manure can only be processed four or five months out of the year because of moisture and cool weather.

TABLE 4. Results of feeding CLW to breeder crossbred heifers.

Treatment	Gain per day for 120 days Utah State University, 1981	
	Average Gain/Day	Percent Diff.
	lbs.	
Alfalfa control	.87	—
Composted CLW	1.02	17
Mechanically composted CLW	1.30	49

TABLE 9. Effect of feeding processed cage layer waste on feed cost¹ per dozen eggs, 1982-83.

Waste Level	Feed/ Doz Eggs	Est. Feed Cost/Doz. Eggs		
		Value allowed for waste 0	1¢/#	4¢/#
	%	lbs.	¢/doz	4¢/#
0	3.43	26.8	26.8	26.8
5	3.62	27.1	27.3	27.8
10	3.743	26.8	27.2	28.3
15	3.89	26.6	27.2	29.0

¹Ingredient cost only.**TABLE 5. Results of feeding mechanically composted layer waste (MCLW) to broiler chicks.**

0-3 Weeks, USU, 1981				
Diet	Replications	Gain	Feed/Gain	Difference in Feed/Gain
Basal	4 × 5	465	1.61	
+5% MCLW	3 × 5	477	1.65	+2.5
+10% MCLW	3 × 5	465	1.65	+2.5

TABLE 6. Results of feeding MCLW to broiler chicks.

3-6 Weeks, USU, 1981						
Diet	Gain		Feed/Gain		Difference in Feed/Gain	
	Exp. 1	Exp. 2	Exp. 1	Exp. 2	Exp. 1	Exp. 2
	gm	gm				
Basal	1030	1084	2.06	2.22		
+5% MCLW	1079	1042	2.13	2.36	+3.4	+6.3
+10% MCLW	1082	1114	2.16	2.36	+4.0	+6.3

TABLE 7. Results of feeding mechanically composted cage layer waste and two protein levels on egg production,¹ 1981-82.

Parameters Measured	15% Protein	+10% MCLW	17% Protein	+10% MCLW
Hen-day production, %	85.8	86.4	85.0	85.2
Mortality, %	7.5	6.0	7.8	6.7
Feed consumption, g/day	110.7	118.8	112.0	119.5
Feed/doz. eggs, lbs/doz.	3.41	3.63	3.48	3.70
Body weight, g	1764.	1856.	1858.	1921.
Egg weight, g	60.3	62.6	61.0	63.5
Egg specific gravity	1.0747	1.0744	1.0749	1.0747

TABLE 8. Results of feeding different levels of mechanically composted cage layer waste on egg production,¹ 1982-83.

Parameters Measured	Level of Waste			
	0	5%	10%	15%
Hen-day production, %	82.5	81.7	83.6	82.3
Mortality, %	4.4	2.5	6.1	2.6
Feed consumption, g/day	107.1	112.0	118.3	121.1
Feed/doz. eggs, lbs/doz.	3.43	3.62	3.74	3.89
Body weight, g	1743.	1764.	1838.	1744.
Egg weight, g	61.0	62.4	63.7	61.8
Egg specific gravity	1.0796	1.0797	1.0792	1.0796

The other method is a mechanically composted method. The basic machine and procedure for mechanical composting was developed by a group in Michigan about five years ago. They have patented their process but have had some problems in getting the process to work under varied field conditions. About four years ago they gave Utah State University a small machine to conduct experiments. The objective was to produce composted cage layer waste for feeding trials. We have modified their procedure and made some major improvements. We are now able to compost fresh cage layer waste into a dry product that would be suitable as a feed ingredient. The mechanical composting process requires about thirty hours for complete processing. The process appears to have commercial applications, when the moisture can be adjusted to about 46 to 48 percent and the air flow and temperature are controlled. The high moisture content of cage layer waste is still the limiting factor for broad application.

Nutritional Values

Animal wastes have nutritional value. Table 1 shows the composition of poultry wastes. Many variables in collection and harvesting of manure, such as length of time between collection and conditions within the house, affect nutritional values, especially the nitrogen content. Nitrogen is reduced by about fifty percent in a thirteen-week accumulation of waste (see Table 2). The mineral content is relatively stable. The waste that has been used in the feeding trials is defined as fresh material (three to seven day collection). When manure is harvested at this interval, the odors are minimal and it doesn't appear to be as corrosive as had been expected. A three-to-four day cleaning cycle should not allow flies to reproduce even in the summer and would help in a fly control program.

Many workers have shown that dehydrated poultry waste has value for feeding to ruminants, which can metabolize the non-protein nitrogen in waste such as uric acid. Chickens excrete nitrogen in the form of uric acid. Workers have shown that uric acid is more efficiently used than urea by ruminant animals.

Naturally composted cage layer waste (CLW) and mechanically composted cage layer waste (MCLW) were evaluated in an experiment utilizing beef replacement heifers. Fifteen head of crossbred heifers averaging 585 pounds were randomly divided into three treatment groups of

equal weight. Treatments consisted of (1) control (alfalfa), (2) composted cage layer waste (CLW), and (3) mechanically composted cage layer waste (MCLW).

Diets consisted of corn silage and three pounds of chopped alfalfa hay for the control group, with composted cage layer waste (CLW) and mechanically composted cage layer waste (MCLW) replacing alfalfa hay for treatments two and three. The diets were formulated to give daily gains of 1-1½ lbs. per day, per head. The cattle were fed once daily in the morning. The corn silage was fed first, alfalfa and CLW and MCLW were then sprinkled on top and mixed with silage. The trial was conducted for 120 days with the cattle being weighed every thirty days.

Results of the feeding trial, by thirty-day intervals, are shown in Table 3. During the first month of the trial, the cattle fed the corn silage-alfalfa mix outgained those fed either CLW or the MCLW. This would indicate that the cattle must adjust to eating these products. In observing the cattle, it was noted that the cattle would sort out the CLW and the MCLW even after careful mixing. Heifers receiving the CLW were the slowest to accept the experimental diet.

By the time the trial was concluded, the cattle on CLW and MCLW were outgaining those on the alfalfa-corn silage diet. Overall gains for the 120-day feeding period are shown in Table 4. This table shows that the cattle fed MCLW gained more weight, followed by CLW. The cattle fed corn silage-alfalfa mixture had the slowest rate of gain.

In another study with Howlett Farms, different objectives were approached: (1) to determine if the naturally composted cage layer waste could be made into a protein range block and (2) to observe the effects of feeding composted waste as a supplement for cow-calf herd. Blocks were made containing 10, 20, and 30 percent CLW. Under the conditions used in manufacturing the blocks, the thirty percent level was about as high as could have been incorporated. Two tons of each level were manufactured and the finished product was returned to Howlett Farms for testing of palatability and performance in January of 1981. There were no observable differences in acceptance by range beef cow of the blocks, thirty percent was just as acceptable as ten percent. The animals were on a poor quality range. The cows consumed about one and one-half pounds of the block per day. The feed supplement blocks were replaced in March with a mixture of barley, composted cage layer waste, and salt. This

was fed free choice in mash form and was readily accepted by the cows. It was necessary to leave the cows on a range consisting of dry wheat grass (*Bromus tectorum*) after the initial spring growth was gone. The range was extremely poor. They decided to feed the cows the same supplement they had been receiving during the winter. Figures 1-3 will best show the results. Each pair of cows and calves consumed about 1½ to 2½ pounds per day depending on the condition of the range. The calves were born during March, April, and May. They were weaned November 19, the steers averaged 505 pounds and the heifers 450 pounds. There were no health problems traceable to feeding poultry waste. The cows were in very good condition and about 20 out of 100 cows calved in December. In previous years, prior to supplementing, the range calves weighed about 350 pounds at weaning and the calves were two months older, also the cows were very thin. These were the same cows, kept on the same range and basically under the same circumstances as the year that the supplement was fed.

The real test of a product such as composted poultry waste is if it can be fed to young fast growing broiler type chicks without health or negative problems. Tests have been conducted with the feeding of MCLW to broilers. Some adjustment was made in the diet for protein and minerals, but no allowance was made in the diet for the reduction of energy due to addition of poultry waste. The results are summarized in Tables 5 and 6. The results from 0 to three weeks (Table 5) indicated that rate of gain was not reduced by the addition of five or ten percent MCLW; about three percent more feed was required per unit of gain. The results from three to six weeks (Table 6) show that rate of gain was not reduced by addition of either five or ten percent MCLW; about five percent more feed was required per unit of gain. There were no apparent negative health problems that could be associated with feeding MCLW to young birds.

Two experiments have been conducted with feeding MCLW to egg producing type chickens to determine its effect on the health of the bird as well as other production parameters. The first experiment in 1981-82 consisted of feeding ten percent MCLW in diets containing two protein levels. Adjustments in calcium and protein content were made but no allowance for the difference in energy content was made. The effective calculated protein level in MCLW used was ten percent. The second experiment in 1982-83 consisted

of feeding zero, five, ten, and fifteen percent MCLW. The results are shown in Tables 7 and 8. There were no apparent negative health problems associated with feeding of MCLW. Feeding up to fifteen percent waste did not reduce egg production or increase mortality. The egg (size) weight increased in groups receiving the five and ten percent MCLW levels but not in groups receiving the fifteen percent level. The body weight of the birds increased also at the five and ten percent levels but not in the birds receiving the highest level. The feed consumption increased in all treatments receiving MCLW. Feed consumption showed a significant linear increase as the level of waste increased.

The influence of MCLW on feed costs to produce eggs is given in Table 9 from the 1982-83 experimental results. The feed costs of producing a dozen eggs are shown with a given value of waste from zero, one, or four cents per pound. Even with zero value given to MCLW, the addition of waste to the diet has marginal value. If a value of one or more cents per pound of waste is used, there is added cost to produce a dozen eggs. Therefore, there is no reduction in costs of producing eggs with the usage of MCLW in diets of hens producing eggs.

In summary, cage layer waste can be processed into a feed ingredient that can be utilized by the ruminant animal more efficiently than poultry. There were no apparent health problems from feeding processed waste to ruminant animals or to poultry. There must be an adjustment period for ruminant animals to adjust to diets containing waste. There appears to be an economic opportunity to supplement low quality ranges with processed cage layer wastes in various forms. Cage layer waste recovery is still limited by the high moisture content of the natural product.

REFERENCES

- Anthony, W. B., 1980. Waste Refeeding: State-of-the-Art. From: *Livestock Waste: A Renewable Resource*, The Proceedings of the Fourth International Symposium on Livestock Wastes, p. 61.
- Bhattacharya, A. N., and Taylor, J. C., 1975. *Recycling Animal Waste as a Feedstuff: A Review*. *J. Animal Science*; 41, 1438-1457.

ABOUT THE AUTHORS

Jay O. Anderson, professor, ADVS Dept., retired.

Donald C. Dobson, associate professor, ADVS Dept.; State Extension Poultry Specialist for Utah.

Norris J. Stenquist, professor, ADVS Dept.; State Extension Livestock Specialist for Utah.

PANTOTHENIC ACID

IN THE HUMAN DIET

Pantothenic acid, a B vitamin, is important for its biochemical roles as a structural component of coenzyme A (CoA) and acyl carrier protein (ACP). These pantothenate derivatives transfer acyl groups through the terminal sulfhydryl ends by forming thiol esters in more than 70 different reactions. Both CoA and ACP mediate the energy metabolism of carbohydrate, lipids, and amino acids. They are also involved in the biosynthesis of such vital compounds as cholesterol, bile acids, acetylcholine, ketone bodies, and porphyrines.

Despite its important biochemical roles, pantothenic acid nutrition in human beings has been rarely evaluated for several reasons. First, there has been a lack of interest concerning pantothenic acid requirements in humans. Since pantothenic acid is ubiquitous in nature, an overt dietary deficiency has not been clinically recognized in man except when a metabolic antagonist, omega-methyl pantothenic acid, was administered (Hodges et al., 1959) or when a pantothenic acid free semi-synthetic diet was given for 10 weeks (Fry et al., 1976). Second, the methodology available for determining pantothenic acid has been very tedious and time consuming, and comparison of results from different laboratories has been inconsistent. Third, means of detecting and evaluating a pantothenic acid deficiency are limited. Since CoA, an active form of pantothenic acid in tissue, plays vital roles in so many different enzymatic reactions, it has not been easy to isolate a single metabolic step than can reflect pantothenic acid nutritional status.

The symptoms of pantothenic acid deficiency induced experimentally in human beings were wide spread. Neuromuscular, cardiovascular, gastrointestinal, immunologic and psychiatric alterations occurred. Disturbed biochemical functions included acetylation, steroid hormone secretion and failure of ACTH to induce eosinopenia. Deficiency symptoms have also been observed in animals (Hurley et al., 1965). These included infertility, abortion, frequent neonatal death, retarded growth rates in young animals, sudden death, abnormalities in skin, hair and feathers,

neuromuscular disorders, gastrointestinal malfunction and adrenal cortical failures.

The extent to which suspected synthesis of intestinal microflora contributes to body needs is unknown. The result of balance studies indicated urinary plus fecal pantothenic acid excretion slightly exceeded the dietary intake but was inconclusive (Gardner et al., 1943).

Concerns About Pantothenic Acid in Human Diet

Human nutritionists today are concerned about the nutritional status of this vitamin for several reasons. First, the Food and Nutrition Board of the National Academy of Sciences and National Research Council for the first time in 1980 recommended an Estimated Safe and Adequate Daily Dietary Intake for pantothenic acid. They suggested an intake of 4-7 mg a day for adults and an undefined "a higher" amount for pregnant and lactating women. Estimated adequate intakes for other age groups are based on their proportional energy needs. More complete scientific data on the human requirement is required so that the tentatively suggested intake can be further refined and suggested as an RDA. Second, food supply patterns have changed. Americans today consume more refined and processed foods which contain substantially less pantothenic acid than the original foods. We have more available formulated foods, food analogs and vitamin and mineral supplements which may increase the risk of deficiency and toxicity. Pantothenic acid loss during processing is significant and can be as high as 70 percent in frozen meat and 80 percent in canned legumes (Schroeder, 1971). Loss of the vitamin in dairy products during processing and storage is about 30-35 percent. Pantothenic acid content of breads and cereals is reduced to 50 percent of the original content when it is milled to 70 percent extraction flour. The vitamin is stable in neutral solution but is readily destroyed by heat at either alkali or acid. Third, the food consumption pattern has been changed with increased intake of sugar, fat, and alcohol which contain neg-

ligible amounts of pantothenic acid and high calories. Simultaneously the public has been limiting their energy intakes rather than changing food consumption patterns.

Pantothenic Acid Status of Elderly Utahn Population

Pantothenic acid status of human beings has been commonly evaluated by use of data on estimated dietary intake and the vitamin levels in blood and urinary excretion. Yet researchers in the area had to surmount many existing problems including tedious and less accurate methodology available, lack of data on pantothenic acid content in foods and insensitive indexes for nutritional status. Our laboratory is one of the few labs in the world which are actively involved in the research on pantothenic acid. We have developed a radioimmunoassay (RIA) for pantothenic acid (Wyse et al., 1979), isolated an enzyme, pantetheinase, that specifically releases free pantothenic acid from bound forms (Wittwer et al., 1982) and extended the determination of the pantothenate content in foods (Walsh et al., 1981). With the new methods and compiled data, we have been able to evaluate pantothenic acid status in Utahns efficiently (Table 1).

In 1981 pantothenic acid nutritional status was studied in the elderly group, 65 years of age or older, both the institutionalized and noninstitutionalized, in Utah by Srinivasan et al. (1981). The study was conducted because nutritional vulnerability of elderly individuals had been widely recognized and the group had never been assessed for their pantothenic acid status. In the study, each subject participated by donating a fasting blood sample, a 24-hour urine specimen and a food consumption record along with food samples for a week. Radioimmunoassay was used for analysis of pantothenic acid in all food and biological samples. We found the participants, who were residents of Logan City and neighboring area, had similar values of hematological, anthropometric and dietary parameters as the elderly populations in other studies. The average

(range) dietary intake of pantothenic acid of the elderly population studied was 5.9 mg/day (2.5-9.5 mg/day) with institutionalized and noninstitutionalized subjects having a similar intake of 2.9 mg/1,000 kcal. Institutionalized elderly males and females were consuming 2,237 and 1,962 kcal, respectively while their noninstitutionalized counterparts were consuming 2,201 and 1,887 kcal. Ninety-one percent of the population studied consumed 4 mg of pantothenic acid or more daily from dietary sources. Pantothenic acid from vitamin supplements plus dietary sources ranged from 2.5 to 122.4 mg/day. A pantothenic acid supplement was consumed by 26 percent of the noninstitutionalized and 19 percent of the institutionalized group. Subjects who consumed less than 4 mg/day, which is the lower limit of the Safe and Adequate Daily Dietary Intake, were 9 percent of the population studied.

Blood pantothenic acid values between the noninstitutionalized (537 ± 27.4 ng/ml) and the institutionalized (615 ± 47.3 ng/ml) were comparable. Urinary pantothenic acid excretion of the institutionalized elderly (7.5 ± 1.3 mg/g creatinine) was comparable to the excretion level of the noninstitutionalized subjects (5.9 ± 0.6 mg/g creatinine). Those consuming pantothenic acid supplements had significantly higher excretion levels. In conclusion, we observed a large individual variation in all parameters and no significant difference between the two groups. The mean dietary intake was within the range of Estimated Safe and Adequate Daily Dietary Intake for adults although 9 percent of the subjects consumed less than the lower limit of the recommendation for adults.

Pantothenic Acid Status of Utah Women

In 1982, pregnant and lactating women in Utah were evaluated for their pantothenic acid status (Song, 1983). The study was conducted because no similar research had been reported in this country. Studies on the effects of pantothenic acid deficiency during pregnancy in rats and in guinea pigs reported a variety of complications and defects in both mothers and litters.

Subjects in the study participated longitudinally at three stages beginning with the third trimester of pregnancy and until 3 months postpartum. Data were collected on dietary intake based on reported records, blood and plasma levels and urinary excretion of pantothenic acid. The dietary record was analyzed by use of a data tape named NUTREDFO developed by Utah State University with a contract with USDA. Daily mean pantothenic acid intake of the nutritionally high risk group was 5.6 mg/day with an average of 2.75 mg per 1000 kcal (Table 2). Interestingly the pantothenic acid intake of pregnant/lactating subjects was not significantly different from 4.8 ± 1.6 mg/day of non-pregnant/non-lactating women. Pantothenic acid intake during pregnancy was not significantly different from that during lactation. This suggests that women in general do not change their eating habits during pregnancy nor during lactation. The dietary pantothenic acid intake per 1000 kcal energy consumption of the women (2.75 mg/1000 kcal) was comparable with 2.9 mg/1000 kcal of the elderly population in Utah. Of the total participants in the study, an average of 26 percent consumed less than 4 mg of pantothenic acid a day, which is the lower limit of the recommendation for normal healthy adults and 23 percent had more than 7 mg/day, which is tentatively suggested intake for the nutritionally high risk group. A few individuals consumed large quantities of pantothenic acid supplements, ranging from 3.3-102 mg/day, often as brewer's yeast. All of the pregnant women consumed prenatal multivitamins and mineral supplements but none contained more than 1 mg pantothenic acid per capsule.

Dietary intakes of macronutrients and energy did not differ from those of national report (Hanes, 1971-74). The daily mean intake of most vitamin and minerals for which RDA are established were above the standards except for folic acid, magnesium, iron, and zinc. These findings are supported by other studies.

Blood levels of the pregnant/lactating women were significantly lower than those of the control women (Table 3). Interest-

ingly, we observed the depressed blood vitamin level during pregnancy was significantly increased during the postpartum period from that during pregnancy. This may be explained by hemodilution or increased needs of the vitamin during the third trimester of pregnancy as observed in a guinea pig study (Hurley et al., 1965). The fasting plasma pantothenic acid levels did not significantly differ between the groups and among the stages. This result suggests that the vitamin level in the fasting plasma does not sensitively reflect human nutritional status.

Urinary pantothenic acid excretion of pregnant women in this study did not differ from that of the non-pregnant/non-lactating women. As expected, the mean urinary pantothenic acid excretion of the subjects who consumed pantothenic acid supplements were significantly higher (12.12 ± 5.80 mg/day) than that of the unsupplemented participants. We suggest, based on the marginally lower dietary pantothenic acid intake and lower blood pantothenic acid level of pregnant women in the study, that pregnant and lactating women in Utah need to consume more pantothenic acid. This may be achieved by a very careful food selection or by an increased caloric intake, if desirable. Supplementation with the vitamin, however, may not be necessary for the Utahans who have easy access to abundant fresh fruits and vegetables and whole wheat products. Pantothenic acid preparations and analogs (e.g., pantoic acid and "vitamin B₅") which are commonly available in health food stores should not be recommended. Their bioavailability and function are unproven.

Pantothenic Acid Content in Human Milk

The proportion of breast-fed infants in Utah is generally known to be higher than the national average. In fact, 26 subjects of the total 29 pregnant women in the above-mentioned study chose to nurse their infants when no influence was given by the researcher as to the infant feeding method.

This study was conducted because there were only a few reports on the pan-

tothenic acid content in human milk, the majority of which, however, was completed abroad and others were reported in the United States more than 30 years ago. In our study (Song et al., 1984), human milk samples were collected at the beginning (fore milk) and at the end (hind milk) of the first feeding of the day from the 26 nursing subjects who had delivered full-term infants at 2-week and at 3-month postpartum.

On the other hand, milk samples from mothers who had delivered preterm infants were collected by the subjects at their home and reached to the researcher through the University of Utah Medical Center.

Pantothenic acid content of the milk samples was analyzed by radioimmunoassay after an extensive validation of sample preparation techniques. We found in the study that preterm milk contained a significantly higher amount of pantothenic acid than full-term milk (Table 4). No significant change was observed in pantothenic acid content within a feeding or with the progress of nursing in both groups. Although a large individual variation was observed, the pantothenic acid content in human milk was moderately ($r=0.151$) correlated with maternal dietary intake and with the vitamin levels in maternal circulation (blood, $r=0.21$; plasma, $r=0.23$) and with urinary excretion ($r=0.57$). Pantothenic acid supplementation resulted in a higher level of the vitamin in milk even though the sample size was too small for statistical significance.

Assuming that an infant receives approximately 850 ml of milk a day during the first three months of life, the amount of the vitamin consumed by the infants of unsupplemented mother in this study would be equivalent to 2.2 mg/day. This amount of intake is slightly higher than the Estimated Safe and Adequate Daily Dietary Intake of 2.0 mg/day suggested for the group. The pantothenic acid content of human milk from term mothers taking no supplement in this study (2.56 ug/ml) is slightly higher per volume than the minimal requirement of the Infant Formula Act of 1980 (2.0 ug/ml).

Interestingly, we also found the level of pantothenic acid in human milk is 5 times higher than that in whole blood and 23 times higher than those in maternal plasma. Similar phenomena of concentrating vitamins by the mammary gland have been observed. The ratio, however, was much larger for pantothenic acid than for other water-soluble vitamins.

One of our on-going research projects is to evaluate the pantothenic acid nutri-

TABLE 1. Dietary intake, blood level and urinary excretion of pantothenic acid in Utah elderly population.

Group	Number of Analyses	Intake (mg/day)	Whole Blood Level (mg/ml)	Urinary Excretion (mg/g creatinine)
Non-institutionalized pantothenate				
supplemented	17	22.7±6.4	557.0±121.0	13.7±2.3*
unsupplemented	48	5.8±0.2	533.0± 22.5	5.9±0.6
Institutionalized pantothenate				
supplemented	5	23.6±2.2	721.0±108.1	15.5±4.6
unsupplemented	21	5.9±0.3	583.0± 51.9	7.5±1.3

Adapted from Srinivasan et al. (1981).

Mean ± Standard Error of Mean.

*Supplemented vs. unsupplemented noninstitutionalized $p < 0.01$.

TABLE 2. Estimated daily nutritional intakes of pregnant, lactating and control women.

Group Observation (n)*	Pregnant n = 26	Lactating n = 46	Control n = 47
Energy (kcal)	2072 ± 467†	2014 ± 620	1800 ± 828
Protein (gm.)	81 ± 19	79 ± 26	71 ± 23
Fat (gm.)	86 ± 26	92 ± 30	73 ± 28
Carbohydrate (gm.)	252 ± 58	241 ± 70	216 ± 138
Calcium (mg.)	1390 ± 750	1243 ± 595	965 ± 390
Magnesium (mg.)‡	364 ± 120	317 ± 101	313 ± 162
Phosphorus (mg.)	1522 ± 487	1446 ± 563	1273 ± 407
Vitamin A (I.U.)	10197 ± 5894	9627 ± 5206	9839 ± 8169
Thiamin (mg.)	2.6± 1.3	2.1± 1.1	1.4± 0.8
Riboflavin (mg.)	3.4± 1.5	2.8± 1.4	2.0± 1.2
Niacin (mg.)	97 ± 107	70 ± 90	58 ± 52
Pyridoxine (mg.)	3.7± 3.0	3.0± 2.7	1.9± 1.2
Vitamin B ₁₂ (g.)	9.3± 6.9	7.5± 5.7	5.8± 9.9
Ascorbic acid (mg.)	238 ± 91	199 ± 129	161 ± 106
Folic acid (g.)‡	704 ± 488	532 ± 419	311 ± 165
Potassium (mg.)	3206 ± 1021	2925 ± 1181	2592 ± 1157
Iron (mg.)‡	33 ± 29	26 ± 24	17 ± 12
Sodium (mg.)	2769 ± 1629	2251 ± 850	2465 ± 1133
Zinc (mg.)‡	13 ± 6	12 ± 5	10 ± 3
Pantothenic acid (mg.)	5.3± 1.7	5.9± 2.0(n=46)* 31.1± 36.9(n=6)#	4.8± 1.6(n=47)* 55.1± 81.0(n=4)#
	5.3± 1.7	8.8± 11.6(n=52)§	9.7(n=51)§

Adapted from Song (1983).

*Subjects taking no or less than 1.0 mg supplemental pantothenic acid daily (unsupplemented group); observation number (n).

†Mean ± standard deviation.

‡Dietary taking more than 1.0 mg supplemental pantothenic acid daily (supplemented group).

§Mean intake of all subjects in the group (both supplemented and unsupplemented groups).

TABLE 3. Mean pantothenic acid levels in blood, plasma, and urinary excretion of subjects taking no pantothenic acid supplement.

Group	Phase I	Phase II	Phase III	Average
Control				
blood (ng/ml)	534.21±20.60*	525.19±19.79	516.97±20.69	525.46±12.01
plasma (ng/ml)	117.51± 6.20	112.68± 5.96	103.81± 6.23	111.33± 3.62
urine (ng/day)	3.00± 0.58	—	2.13± 0.40	2.59± 0.17
Experimental (pregnant/lactating women)				
blood (ng/ml)	404.16±15.81†	444.81±17.36†	451.26±17.11†	433.41± 9.96†
plasma (ng/ml)	108.93± 4.76	112.91± 5.23	102.46± 5.15	108.10± 3.00
urine (ng/day)	3.26± 0.44	—	2.65± 0.48	3.29± 0.23

Adapted from Song (1983).

*Mean ± standard deviation.

†Significantly ($p < 0.05$) lower than those of the control group.

—Samples were not collected.

Phase I, II, III ascribe the 3rd trimester of pregnancy, 2 week postpartum and 3 month postpartum of experimental group.

Control: non-pregnant and non-lactating women.

Experimental: pregnant women who participated at the three phases.

tional status of Utah adolescents. Approximately one-hundred girls (ages 13-17) and boys (ages 14-21) will be recruited through high schools in Logan and surrounding areas. The volunteers will be oriented by a researcher, Brenda Eissenstat, for the research plan and their responsibilities. Pantothenic acid intake will be calculated from a 4-day dietary record, and the vitamin levels will be determined in a fasted blood and 2-day's pooled urine samples. This research project is planned to be completed by summer, 1985.

The foods high in pantothenic acid content are listed in Table 5. In general, high protein foods, whole wheat grain, fresh vegetables, and fruits are good sources of the vitamin.

ABOUT THE AUTHORS

Won O. Song received a PhD at USU in Nutrition and Food Sciences at USU in 1984 working in the lab for one year as a post-doctoral fellow. She is continuing research in the area of pantothenic acid at Michigan State University.

Bonita W. Wyse is the Acting Dean for the College of Family Life and professor of Nutrition and Food Sciences and director of the Medical Dietetics Program, Department of Nutrition and Food Sciences. She has had a continuing interest in communicating nutrition education for the public and has conducted a research program in that area.

R. Gaurth Hansen is professor in the Departments of Nutrition and Food Sciences and Chemistry/Biochemistry. He received his PhD from the University of Wisconsin in Biochemistry. His current research is with pantothenic acid and nutrition education.

REFERENCES

- Fry, P. C., Fox, H. M. and Tao, H. G. Metabolic response to a pantothenic acid deficient diet in humans. *J. Nutr. Sci. Vitaminol.* 22:339-346, 1976.
- Gardner, J., Neal, A. L., Peterson, W. H. and Parson, H. T. Biotin, pantothenic acid and riboflavin balances of young women on a milk diet. *J. Am. Dietet. Assoc.* 68:684, 1943.
- Hodges, R. E., Bean, W. B., Ohlson, M. A. and Bleiler, R. Human pantothenic acid deficiency produced by omega-methyl pantothenic acid. *J. Clin. Inv.* 38:1421-1425, 1959.
- Hurley, L. S., Volkert, N. E. and Eichner, J. T. Pantothenic acid deficiency in pregnant and non-pregnant guinea pigs, with special reference to effects on the fetus. *J. Nutr.* 86:201-208, 1965.
- Schroeder, H. A. Loss of vitamins and trace minerals resulting from processing and preservation of food. *Am. J. Clin. Nutr.* 24:562-573, 1971.
- Song, W. O. Longitudinal pantothenic acid status of pregnant and lactating women. Ph.D. dissertation. Utah State University, 1983.
- Song, W. O., Chan, G. M., Wyse, B. W. and Hansen, R. G. Effect of pantothenic acid status on the content of the vitamin in human milk. *Am. J. Clin. Nutr.* (in press).
- Srinivasan, V., Christensen, N., Wyse, B. W. and Hansen, R. G. Pantothenic acid nutritional status in the elderly—institutionalized and non-institutionalized. *Am. J. Clin. Nutr.* 34:1736-1742, 1981.
- Walsh, J. H., Wyse, B. W. and Hansen, R. G. Pantothenic acid content of 75 processed and cooked foods. *J. Am. Dietet. Assoc.* 78:140-144, 1981.
- Wittwer, C., Wyse, B. and Hansen, R. G. Assay of the enzymatic hydrolysis of pantothenic acid. *Anal. Biochem.* 122:213-222, 1982.
- Wyse, B. W., Wittwer, C. and Hansen, R. G. Radioimmunoassay for pantothenic acid in blood and other tissues. *Clin. Chem.* 25:108-111, 1979.

TABLE 4. Mean pantothenic acid content of term and preterm human milk.

Group	Collection Period	Number	Fore Milk (Range) g/ml	Hind Milk (Range) g/ml	Combined g/ml
Term	2 wk postpartum	22	2.73±0.61* (1.75-4.05)	2.40±0.58 (0.95-3.70)	2.57±0.60
	12 wk postpartum	24	2.54±0.72 (1.00-3.71)	2.55±0.73 (1.00-4.84)	2.55±0.73
	Mean		2.64±0.67	2.48±0.66	
Preterm	<40 wk gestational age	14	3.58±1.41 (1.72-7.27)	4.23±1.67 (1.71-6.67)	3.91±1.54†
	>40 wk gestational age	15	3.06±0.35	3.25±0.70	3.16±0.55†
	Mean		3.31±1.01†	3.72±1.26†	

Adapted from Song et al. (1984).

*X ± SD.

†Significantly higher than that of term group (p < 0.05).

TABLE 5. Pantothenic acid contents of foods (per 100 g edible portion).

< 1.0 mg	< 1.0 mg	> 0.4 mg	> 0.4 mg
Avocado, raw	Roe, cod or haddock	Apricots, dried	Crab, cooked, canned
Figs, dried	Lobster	Dates	Lamb
Broccoli, raw	Cheese, blue	Prunes, dried	Pork, fresh, ham
Carrot, dehydrated	Milk, dry		Rabbit
Cauliflower, raw	Ice cream, vanilla	Asparagus, raw, frozen	Beef, lean
Kale, raw	Buttermilk, powder	Collard, frozen	Bran, all kinds
Mushroom, raw, canned	Egg, raw, cooked, dried	Kale, frozen	Spleen, all kinds
Pepper, mature, raw	Green peas, raw	Chicken	
		Pumpkin, canned	Clams
Cashew nuts		Beans, mature	Fish, all kinds
Filberts		Broccoli, frozen	Luncheon meat, canned
Peanuts		Brussel sprouts, raw, frozen	Turkey
Pecans		Cauliflower, frozen	Veal
		Celery, raw	Cheese, Cheddar, Parmesan
Buckwheat flour, dark		Pepper, immature, green	
Oatmeal, dry		Sweet potato	Barley, pearled, light
Heart, all kinds		Almonds, dried	Pretzels
Kidneys, all kinds		Chestnut, fresh	Bread, cracked wheat, rye, whole wheat
Liver, all kinds		Walnuts	Cereals, ready-to-eat, whole wheat
Liverwurst			Pie, custard, pumpkin

Adapted from Orr, M. L., Pantothenic Acid Vitamin B1 and Vitamin B12 in foods. Home Economics Research Report No. 36. USDA, 1969.



Fermented sausage products in which sugars are fermented to lactic acid are growing in popularity in this country due to pepperoni and other ethnic sausages that are being used as pizza toppings or by themselves. The growth of the fermented (dry or semi-dry) sausages market was 3 percent for 1982 partly because of the 9 percent growth in the pizza market according to USDA figures for federally inspected red meat plants. Fermented sausage products offer a potential market for sheep and beef that are produced within this state.

Fermented sausage products have been made traditionally from comminuted beef ingredients with the indigenous microflora of the beef ingredients or "back-slopping" from previous sausage batches providing the culture organisms. More recently, the use of lactic acid bacteria starter cultures has improved fermented sausage quality by producing sausages with fewer batch failures and in a shorter period of time.

Indigenous lactic acid bacteria offer the advantages of having been naturally selected for growth in the meat ingredients being used. Starter cultures can be

added to the sausage formulations to provide a sufficient number of organisms to insure successful, repetitive batch fermentations.

Mutton has the potential of being used more extensively as a fermented sausage ingredient. This meat has a saturated fat which is less subject to rancidity than beef fat and has good economic possibilities as a sausage ingredient. Utilization of mutton as a fermented sausage ingredient would lower the cost, making fermented sausage available to more people and at the same time would command a greater price for mutton than producers are now receiving.

Lamb and mutton consumption has dropped from 3.0 kg per capita (carcass basis) in the 1931-40 years to 0.7 kg per capita at the present time (Field et al., 1983). Possible reasons given for this drop in meat consumption are as follows:

1. price
2. availability
3. perceived high fat and bone to lean ratio
4. the cholesterol content
5. unpleasant experiences with lamb or mutton flavor

6. habit or tradition

7. unacceptance of lamb or mutton flavor by consumers in this country (Crouse, 1983).

Lamb and mutton fat contain both the species related flavor and high levels of saturated fatty acids (Cramer, 1983) which causes the fat to be harder than beef, pork, or poultry fats. Ram sex-related flavors have also been related to the strong lamb or mutton related flavor (Crouse, 1983) and diet contributes to these flavors as well (Field, et al., 1983). Lamb and mutton flavors generally increase with age (Field, et al., 1983).

The lamb/mutton fat may not be a flavor problem in many processed meats due to smoke, spices, and flavorings that can mask specie related flavor. Fermented meats, many of which are highly spiced, also may contain microorganisms that are capable of breaking down the fat and possibly improving flavor due to the lipases (fat splitting enzymes) they contain. Fat from beef and pork can also be substituted for lamb/mutton fat. These ethnic related sausages are currently showing strong growth in this country.

The sheep industry is one of the main agricultural industries within the state. According to 1980 USDA figures, Utah ranked 7th in the nation in the production of sheep and lambs with 507,000 head and 36th in the cattle and calf production with 840,000 head. Using mutton as a sausage ingredient will improve the market value for old ewes within the state and reduce the cost of fermented sausage products. The sheep industry in the state has been depressed for the last two years. A recent local newspaper article epitomizes the plight of Utah's sheep industry by saying that 47 cents/lb for lamb does not cover production costs which amount to 62 to 63 cents/lb. State production figures indicate a 10 percent drop in the state sheep herd between 1980 and 1983. Increased consumption of lamb and mutton in processed meats is a top priority in helping this industry to survive within the state of Utah.

Previous Work

Production of fermented sausages usually involves a fermentation step to permit the production of lactic acid from sugars by lactic acid bacteria and a drying step to remove moisture from the sausage. The traditional or natural fermentation process with indigenous microflora takes from 3 to 7 days, but the more recent starter culture process using frozen concentrates has reduced fermentation times to 15-20 hours (Acton, 1977). Improvements in the natural fermentation process, however, have been made by controlling meat holding times and temperatures and adjusting salt levels to 3 percent which permits natural lactic acid microflora to proliferate (Palumbo et al., 1976).

Indigenous lactic acid microflora in mutton and beef are very likely to be different due to meat composition differences produced by diet dissimilarities. Cultures isolated in the natural fermentation process and commercial starter cultures have the potential of producing and altering flavor related compounds (Bothast et al., 1973). Indigenous lactic acid bacteria may function better than starter cultures in producing lactic acid at a faster rate and yield compounds that enhance sausage flavor.

In 1983, we completed the study on cultivating lactobacilli on beef or mutton using 1.5 or 3.0 percent sodium chloride (NaCl) and 5 or 10°C incubation for 5 or 10 days. Sodium nitrite at the level of 120 mg/kg of meat was added to each of the 32 meat samples. More lactobacilli were cultivated on mutton than beef samples (1×10^7 versus 2×10^4 organisms/gm).

Holding beef samples under anaerobic conditions at 5°C for 10 days with 1.5 percent NaCl was optimal for growing lactobacilli while mutton samples produced more lactic acid bacteria at 10°C for 10 days with 1.5 percent NaCl. Differences were also found for lactobacilli selective media with MRS agar (Difco) producing higher plate counts than LBS agar (BBL).

Fermented sausages offer potential for greater economic return on investments for mutton in either partial or total replacement of beef as the primary sausage ingredient. Mutton has been used successfully with beef to produce non-fermented sausages such as frankfurters (Marshal, et al., 1977). Fermented sausages have excellent shelf-life because of their lactic acid content and low water activities. Semi-dry sausages require refrigerated storage because of their 45 to 50 percent moisture content while dry fermented sausage with less than 35 percent moisture do not require refrigeration (Acton, 1977). Lactic acid bacteria inhibit food pathogens and indigenous spoilage organisms in meat (Bartholomew and Blumer, 1980) and offer a "built-in" safety factor against food poisoning because of their antagonistic effects.

Lamb and mutton specie related flavors are very closely related and can be used interchangeably. These flavors are thought to be produced by (1) the thermal reactions of adipose tissue precursors and/or (2) the reactions of adipose tissue precursors and muscle thermal degradation products. "Mutton" flavor is produced by the aroma from volatile compounds and the taste is produced by non-volatile compounds. Compounds that appear to be factors in producing mutton flavor are lactones, heterocyclic volatile compounds, mercaptans, organic sulfides and 9 and 10 carbon branched-chain acids. Lean lamb and mutton meats have very little "mutton" flavor which is found mainly in the fat (Cramer, 1983).

Mutton flavor in the adipose tissue can be controlled in the following ways in processed meats:

1. Reducing fat to a level of 10 percent in processed products (Wenham, 1974; Anderson and Gillett, 1974; and Brennan and Mendenhall, 1981).
2. Using spices (Baliga and Madaiah, 1970), smoking, and curing (Ogmundsson and Adalsteinsson, 1979) to mask the "mutton" flavor, and
3. Modifying and/or destroying "mutton" flavor precursor compounds through chemical (e.g., formalin) (Kramlich, et

al., 1973) or microbiological modifications (e.g., lipase or other enzymes).

Objective

The objective of this study was to determine the best methods for producing a mutton/beef fermented sausage.

Methods

Summer sausage is a semi-dry fermented sausage that is usually made from all beef meat ingredients with added starter cultures or indigenous microflora used for the fermentation step.

Previous work in our laboratory had indicated that indigenous lactic acid bacteria microflora could be cultivated more readily by using 1.5 percent NaCl versus 3.0 percent NaCl (table salt) both with 120 ppm sodium nitrite and holding the samples at 10°C (50°F) versus 4°C (40°F) under anaerobic conditions. In this study, we cultivated indigenous lactic acid bacteria by adding 1.5 percent NaCl and 120 ppm sodium nitrite to the meat formulations and then holding the samples aerobically in closed (ovac bags at 10°C (50°F) for 5 or 10 days. The lactic acid bacteria microflora was enumerated by standard plate counts on LBS agar (Lactobacillus Selective agar from BBL) in anaerobic jars (BBL) after incubation for 72 hours at 30°C (86°F).

The frozen concentrated starter cultures for this study were obtained from A.B.C. Research Corporation, Gainesville, FL, 32608. We used *Lactobacillus plantarum* (Lp 18) at a level of 6×10^7 bacteria/gram of meat in the meat formulation for lactic acid production. Their *Micrococcus* starter was added at a level of 2×10^8 organisms/g because its lipase enzyme affects the fat and product flavor.

Three different meat formulations were used, each with the final fat content adjusted to 20 percent. The meat formulations were all mutton, lean mutton (7.5 percent fat) and beef fat (blend), and all beef. The standard raw summer sausage formulation is given in Table 1.

The summer sausage was formulated by adding 1.5 percent NaCl and 120 ppm to the meat initially and holding the samples to cultivate indigenous lactic acid bacteria at 10°C (50°F) or freezing the samples to which starter cultures would be added at -30°C (-22°F) until used. The remaining ingredients were hand mixed with the meat having indigenous microflora or starter cultures were added along with the remaining ingredients to the thawed meat samples. The samples were

TABLE 1. Raw summer sausage formulation:

Ingredient	%
Meat	93.923%
Sodium chloride (table salt)	2.75
Corn syrup solids	1.5
Sucrose (table sugar)	1.0
Ground black pepper	0.23
Ground white pepper	0.17
Garlic powder	0.015
Monosodium glutamate	0.2
Sodium nitrite	0.012
Liquid Smoke	0.2
	100.000%

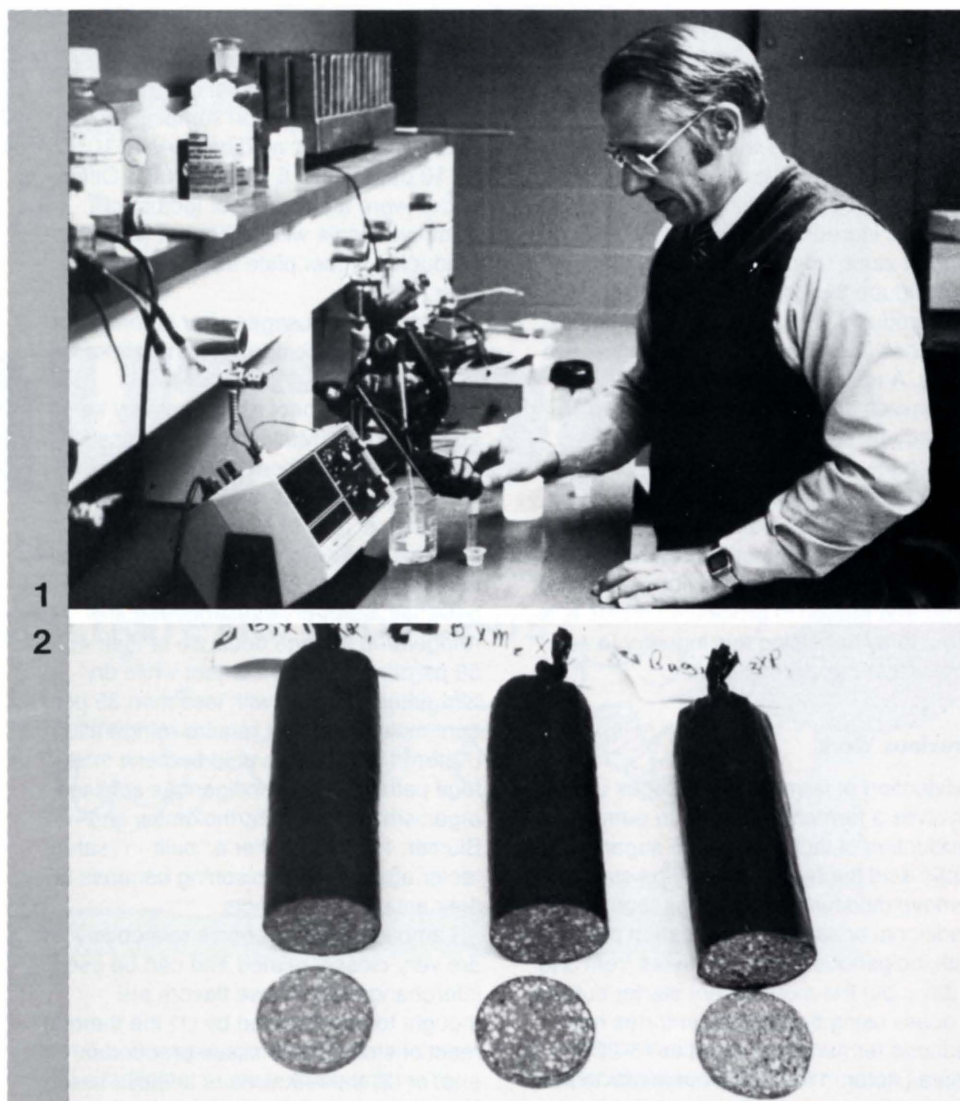
then ground through a meat grinder with a 0.64 cm (1/4") grinding plate to give a final mixing to the sausage prior to stuffing. The different sausages were stuffed into No. 2 Fibrous Mahogany Securex casings from Teepak, Inc., Chicago, IL.

The fermentation step follows formulation and requires that the sausages be held at 32°C (90°F) and 90 percent relative humidity for several hours until the pH of the sausage is between 4.8 to 5.2. We measured the acid production in the sausages by taking pH measurements with a pH meter at different time intervals during the fermentation. Slices approximately 1.27 cm (1/2") thick were cut from the bottom of the sausages, blended with 100 ml (3.38 fl. oz.) of water and then the pH was measured (see Figure 1). The pH measurements can also be made with pH paper or by noting the toughening of product texture that occurs as the meat pH drops below the isoelectric point (5.3-5.5) which is where the meat proteins are least soluble and toughest. The isoelectric point is also where most meat has its poorest water binding capacity and some meat proteins are denatured. When the sausages reached a pH of 5.0 they were removed from the smokehouse and refrigerated until the cooking step. The sausages were cooked in the smokehouse at 68°C (155°F) and 66 percent relative humidity to a final internal temperature of 140°F. The cooked sausages were cooled in a cold water shower for 5 minutes and then covered and held in a 0°C (32°F) cooler.

A consumer preference taste panel was used to evaluate the finished products and appropriate statistical analyses were utilized to analyze the data.

Results and Discussion

Natural lactic acid bacteria were successfully cultivated using 10°C, 1.5 NaCl, and 5 or 10 days in the closed Cryovac bags. LBS place counts were lowest for the all



mutton sausages versus the all beef and mutton/beef blends (Figure 3). The differences between 5 and 10 day samples were most notable for mutton where the 10 day sample had 6.5 times the bacteria of the 5 day samples.

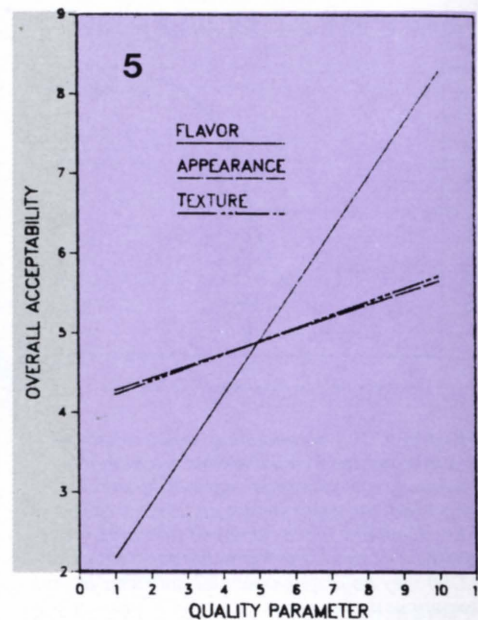
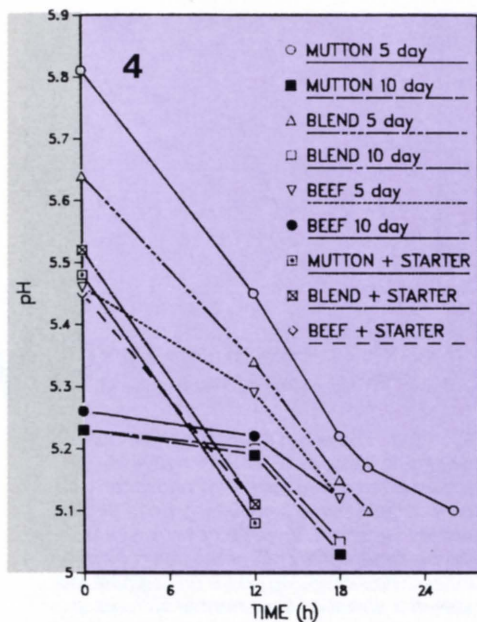
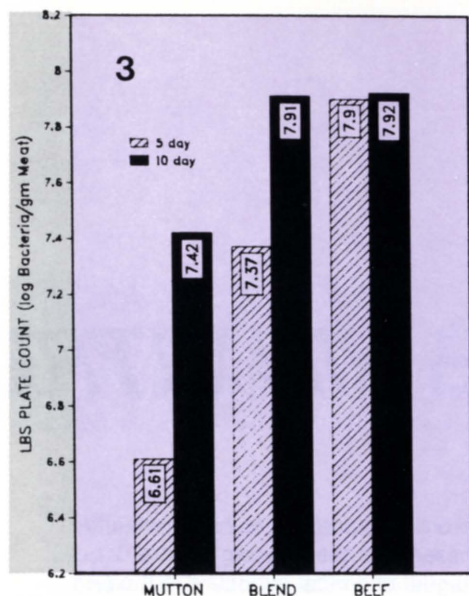
The plate counts are the average of duplicate samples for each treatment. The 5 day samples did not have any off-odors or mold spoilage as did most of the 10 day samples which were held under aerobic conditions. This spoilage problem was not encountered in a previous study because the meat was vacuum packaged (unpublished data).

Because of the spoilage problem encountered with the 10 day samples, we recommend that only the 5 day incubation at 10°C be used for samples held aerobically in closed containers. The sodium nitrite and sodium chloride inhibited the non-lactic acid bacteria microflora for 5 days but not for 10 days incubation.

Definite differences were observed in the fermentation step for the natural bac-

teria versus the starter culture samples (Figure 4). Each of the data points in Figure 4 is an average of duplicate sausage samples. Sausages with starter cultures added had a fermentation time of 12 hours, whereas indigenous lactic acid bacteria samples required 18-26 hours to reach an end point of pH 5.1 possibly due to different acid producing capabilities. The 5-day mutton sausages with indigenous lactic acid bacteria apparently had the longest endpoint fermentation time of 26 hours because these samples also had the lowest level of lactic acid bacteria of 4×10^6 per gram of meat at the start of the fermentation period (see Figure 3).

Samples of the finished summer sausages are pictured in Figure 2 with all mutton on the left, mutton/beef blend in the middle and all beef on the right. Sixteen taste panelists rated four sausage samples for appearance, flavor, texture and overall acceptability on a scale of 1 (dislike extremely) to 10 (like extremely). The taste panel also specified whether the



sample was beef or mutton. The following were sampled: mutton + starter, mutton 5-day, blend 5-day and beef 5-day sausages. The taste panel rated the four sausages as being significantly different ($p = .05$) for appearance, flavor, and texture (Table 2), but overall acceptability was not significant. Mutton + starter and mutton 5-day sausages were rated as having the best appearance and the blend and beef 5-day sausages were rated as having the worst appearance mainly due to fat particle size.

The blend 5-day and mutton + starter sausages had the best flavor and the beef and mutton 5-day sausages the worst. The mutton 5-day sausage had a strong mutton flavor, but the starter culture helped to reduce the mutton flavor in the mutton + starter sample. The beef 5-day sausage had a significantly different texture which was softer and mushier than the other sausages. Overall acceptability was highest for mutton + starter and blend 5-day sausages and lowest for beef 5-day sausage, but these differences were not significant. Flavor has the most effect on overall acceptability while appearance and texture have the same basic effect (Figure 5). Taste panel members only identified the mutton 5-day sample correctly as being mutton ($p = .05$). Ten of 16 panelists identified mutton + starter correctly, only 6 of 16 identified the blend as mutton and 4 of 16 panelists identified the beef 5-day sausage as mutton. The taste panel results indicate that beef fat can replace mutton fat to improve mutton acceptability and that starter cultures will reduce mutton flavor in all mutton sausage.

PHOTO CAPTIONS

FIGURE 1. Dr. Darrell Bartholomew blends sausage sample in water for pH determination.

FIGURE 2. Summer sausage samples shown are all mutton (left), mutton/beef blend (middle), and all beef (right) that taste panelists rated.

FIGURE 3. LBS plate counts of indigenous lactic bacteria by meat formulation.

FIGURE 4. Fermentation of summer sausages with natural microflora or starter cultures to an endpoint pH of 5.1.

FIGURE 5. Interaction of flavor, appearance, and texture with overall acceptability.

TABLE 2. Means of taste panel parameters for summer sausages.

Sample	Appearance	Flavor	Texture	Overall Acceptability
Mutton + Starter	7.3750 A*	6.1563 AB	7.1250 A	6.1875 A
Mutton 5 day	6.9375 AB	4.5625 B	6.7500 A	5.2500 A
Blend 5 day	5.6250 BC	6.3125 A	6.5625 A	6.1875 A
Beef 5 day	5.1875 C	4.6875 B	5.4375 B	4.875 A
LSD	1.3569	1.60666	1.05145	1.37141

*Means with the same letter are not significantly different. Each mean is an average of 16 responses.

Conclusions

1. Starter culture was useful in reducing fermentation time.
2. Lower LBS plate counts were attained on all mutton samples which prolonged fermentation time for the mutton 5 day sample.
3. Indigenous microflora can be cultivated successfully by adding 1.5 percent NaCl and 120 ppm sodium nitrite to the meat and incubating the mixture in a closed container for 5 days.
4. Starter cultures can reduce mutton flavor in fermented sausage.
5. Substituting beef fat for mutton fat and using mutton lean will produce a summer sausage that has no mutton off-flavor.

ABOUT THE AUTHORS

Darrell T. Bartholomew is an assistant professor in the Department of Nutrition and Food Sciences. He teaches classes and conducts research in meat and food processing and meat microbiology.

Bruce L. Woodbury has completed his M.S. degree in the Department of Nutrition and Food Sciences while on leave from the U.S. Army. He is currently serving as a Captain in the Army at Fort Lee, VA.

Chima I. Osuala is a graduate student in the Department of Nutrition and Food Sciences and is working on his M.S. degree.

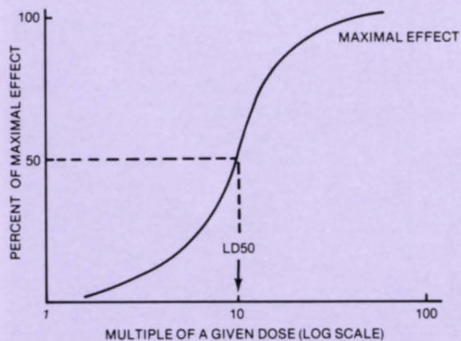


FIGURE 1. A theoretical-log-dose-response curve expected in most animal experiments. Generally a symmetrical sigmoid function is expected, the point shown as LD50 is obtainable if lethality is the observed parameter at different dose levels. A precise estimate of LD50 may need well over 100 animals per experiment.

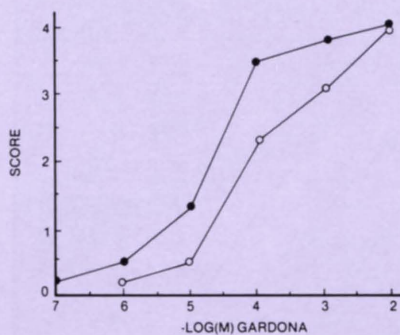
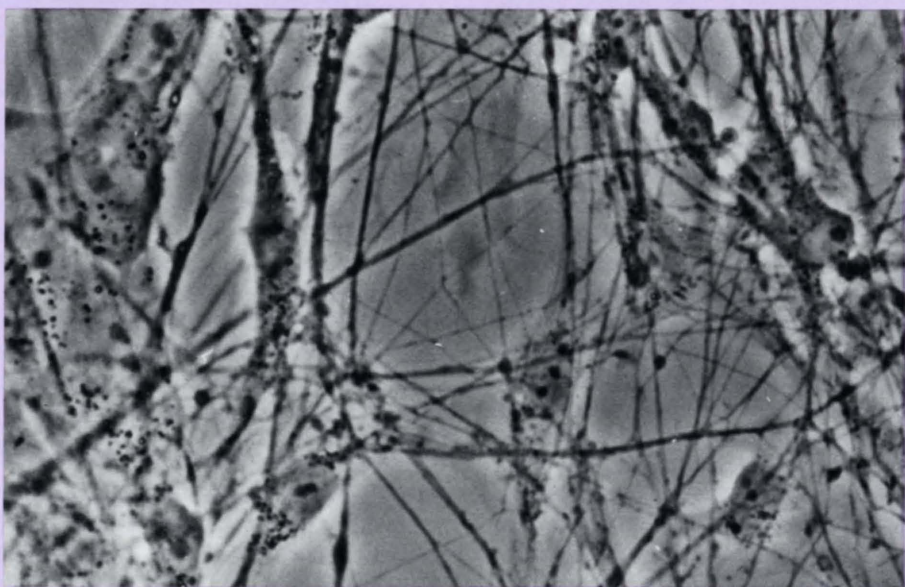
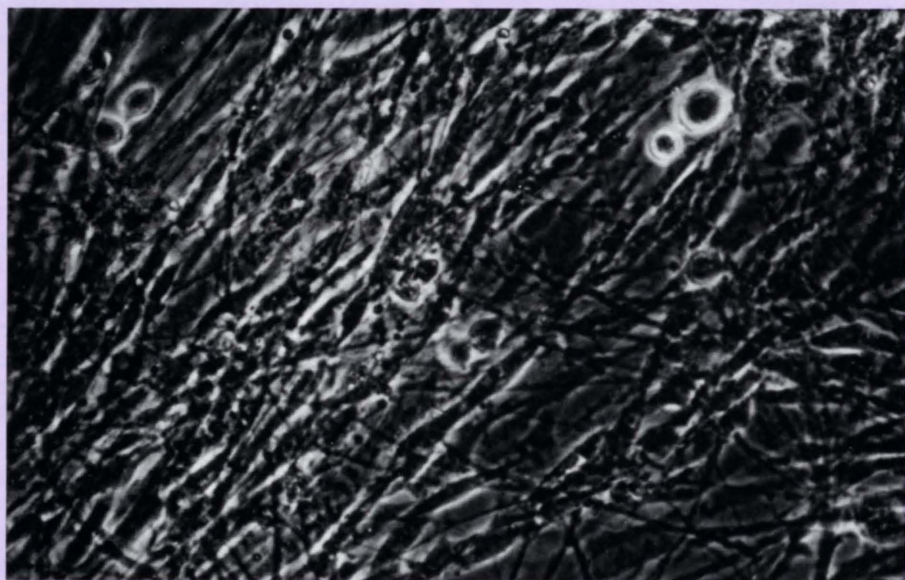


FIGURE 3. The effect of organophosphorus insecticide gardona (2-chloro-1-[2, 4, 5-trichlorophenyl] vinyl dimethyl phosphate) on chick ganglia nerve cell culture. Solid circles indicate the effect on nerve cells, whereas the effect on nueroglia cells is indicated by open circles. The increasing score on ordinate indicates that deviation from normal cultures, a score of 4 being the maximal inhibition of cell growth.



IN VITRO

In order to predict the human health hazards or the economics of a potential chemical exposure, a modern day toxicologist relies largely on data derived from experimental evaluations that involve laboratory animals. (The alternative is to wait for the ill effects to become obvious in exposed people.) Typically, such laboratory investigations require large numbers of experimental animals on which tests are conducted. Commonly used laboratory animals are rats, mice, guinea pigs, hamsters, rabbits, and various species of livestock. To a limited extent cats, dogs, and monkeys are involved. Occasionally tests are conducted on human volunteers, although this practice is gradually diminishing.

All of us, voluntarily or involuntarily, are in daily contact with a large number of chemicals. Pesticides and other chemicals are constantly introduced into our environment to provide a cheaper and better food or to eradicate pests. Mining and metallurgical processes can cause increases in exposure to various toxic metals. Similarly, the manufacturing and even the disposal of natural or synthetic consumer items also can yield health risks. Additionally, breakdown products of the original chemicals may exist for a long time. Long-term chemicals can induce low-level exposure to certain cumulative and irreversible effects (e.g. carcinogenic response). For toxicologists, this can require tedious and thorough testing of the suspect chemicals.

FIGURE 2. (a) A representative phase contract photomicrograph of 3-day old chick ganglia nerve cells, untreated ($\times 3000$). (b) the effect of methylparathion ($1 \times 10^{-5} M$) is illustrated on a similar culture. Note the degenerative effects and less numerous nerve fiber extensions and glial cells ($\times 300$).

TOXICITY EVALUATION

As demand for and costs of animals used in toxicological research soared (Rowan 1979), various scientists began looking for alternatives to experimental laboratory animals. Concern about animal welfare has produced legislations that require a rigid code for housing, care, and ultimate disposal of laboratory animals. In some tests such as an evaluation of LD50 (what dose of a chemical does it take to kill 50 percent of the treated animals in a given period?) hundreds of animals may be needed to provide a statistically precise estimate (see Figure 1). The LD50 value is often required to define the relative toxicity of a chemical and provides a guide for subsequent acute and chronic toxicity studies. The value also serves as a basis for those who must define safeguards to be used against the toxic hazards of a chemical.

Animals are essential in toxicology research. Certain tests can only be conducted in live animals if meaningful data are to be obtained. Several so called "*in vitro*" (non-live-animal) tests have been developed, however, that are indicative of relative toxicity (and hence relative safety) of chemicals. These are particularly useful in investigating the biochemical or molecular mechanisms that underlie toxic effects of such chemicals. Investigators within the Utah State University Toxicology Program have been involved in developing and evaluating such methods. Some of the advantages, drawbacks and results of these tests are summarized in Table 1.

***In Vitro* Techniques Using Tissue or Cell Cultures**

The use of organ, tissue, or cell cultures has been increasing in various biomedical research fields because: a uniform population of cells can be

tested at various exposure levels, and cells derived from a single animal can be employed to complete the whole experiment. Also, some cells will survive in culture over long periods. These are often derived from either embryonic (fetal) organs or from cancerous tissues (tumors). Such cultures not only can be kept alive for a long time but the cells will multiply and produce an infinite supply of material for investigative purposes. In many cases, the data gained from cultured cells can be extrapolated from live organs or animals. The inherent dilemma encountered is relating laboratory animal health effects to humans. Table 2 lists some of the *in vitro* systems that are employed in research.

A few representative USU studies employing various agricultural and environmental chemicals are illustrated as follows.

Chick Dorsal Root Ganglia for Evaluation of Neurotoxic Effects

We have used the sympathetic dorsal root ganglia from chick embryos to evaluate the toxicity of various pesticides and heavy metals. This system provides a mixed-cell, nerve-tissue culture system in which the effects of chemicals can be studied on both nerve fibers and neuroglial cells (Figure 2). The nerve fibers were evaluated for their number, length, and any abnormalities in appearance (such as nodal swelling). The glial cells were scored for their density, size, vacuolization, and general appearance such as spindle form shape or rounding (Watanabe and Sharma, 1975). The effects of the tested chemicals proved to be dose-related and followed a similar pattern as had been predicted for LD50 type effects (Figure 3).

An evaluation of 52 pesticides, including organophosphorus compounds, carbamates, and miscellaneous insecticides, produced interesting results (Sharma and Obersteiner, 1981a). Although the effects in our culture system were dose-related for all compounds, in the case of organophosphate insecticides, no prediction could be made from the cytotoxicity data as to their potential neurotoxicity as had been described in clinical observations. Neither the LD50 nor the extent of cholinesterase inhibition (a well-known mechanism of the toxic effects of these insecticides) were related to the cytotoxicity parameters. In case of the organophosphate insecticides, the cytotoxicity measured in either neuronal cells or neuroglial cells was, however, related to the lipophilic nature of these chemicals. In the case of selected carbamate insecticides, the cytotoxicity index was correlated with cholinesterase inhibition and the lipid solubility of chemicals, but not with the potential to cause lethal effects in animals.

In a different set of experiments, a number of metallic compounds (representing ionizable toxic metallic salts, their metallic oxides and organometallic counterparts) were evaluated in similar, chick sympathetic ganglia cultures (Sharma and Obersteiner, 1981b). In general the oxides were less injurious to the culture system, perhaps due to their limited solubility in the system, which would give them a restricted bioavailability. No relationship was observed between the *in vitro* cytotoxicity and the *in vivo* toxic potential of these compounds. The nerve cell specificity of a number of metallic compounds that has been recorded in animals was not observed in a culture system.

The USU *in vitro* systems appear to be an alternative to using live animals in toxicological research.

Studies Using Bovine Kidney Cell Cultures

We have continued some of the above studies, particularly those with metals, using bovine kidney cells in culture. Metals like mercury, cadmium or vanadium tend to accumulate in kidneys when live animals are exposed to dietary sources contaminated with these elements. Detailed studies in the culture system using such metals and established bovine kidney cells (MDBK cell line) indicated that the cytotoxicity can be somewhat dependent on the intracellular accumulation of metal ions, rather than on the exposure concentrations. Of several biochemical parameters tested, the inhibition of K^+ -dependent phosphatase (a membrane-bound enzyme) and the intracellular induction of glutathione were common effects of all the three metals. Typical morphological alterations were also produced as indicated in Figure 4.

Recently we have studied the effects on a similar system of several mycotoxins (products of fungal contaminants) that are commonly found in foods. A comparison of effects on an established (MDBK) cell line to those seen with cultures of primary origin (bovine fetal kidney) revealed that the perpetuated (established line) cells are deficient in activating mechanisms. Toxicity of aflatoxin B₁ was greater in primary cultures than in the MDBK cells. This chemical is known to be altered by cellular enzymes to its toxic metabolites, which bind to various cell organelles and produce the toxic effect.

An illustration of mycotoxin-induced cytotoxicity in cultured cell line is represented in Figure 5.

Models for Organogenesis

In certain culture systems, cells aggregate and provide cell-cell interactions that are representative of

TABLE 1. Advantages and disadvantages of using animals vs. *in vitro* systems in toxicology evaluation.

	Intact animal	<i>In vitro</i> system
Advantages	Absorption, distribution, metabolism excretion of toxicant can be studied	Specific target organs of tissues can be studied
	Acute and chronic effects of toxicants can be studied	Studies of toxic mechanisms of chemicals on cellular and molecular level are possible
	Behavioral, hormonal and immunological changes can be observed	Inexpensive and rapid
Disadvantages	Very large numbers of living animals are often needed	General systemic effects of chemicals or their effects on tissues or functions cannot be maintained <i>in vitro</i> (e.g. effects on the brain, sight, hearing) and are difficult to determine
	Possibility of inflicting pain on animals	
	Time consuming and expensive	The dose effective to humans is difficult to determine

TABLE 2. Various *in vitro* models used in research

Systems	Types of Assay	End Points Observed
Perfused organs	Whole organs (liver, kidney, heart) are maintained <i>in vitro</i> for short periods	Absorption, distribution and metabolism of chemicals
Tissue slices	Sliced or homogenized tissues are maintained <i>in vitro</i> for short periods	Metabolism of chemicals Enzymes and chemical interactions
Organ cultures	Organs such as liver, kidney, pancreas, adrenal gland, spleen, heart, stomach epithelium, stomach smooth muscle, bladder, skin, lung and oviduct are maintained in a nutrient medium for 24 hrs or longer	O ₂ consumption Glucose uptake/release Pyruvate release Lactate release Nitrogenous excretion Cell proliferation
Cell cultures	Cells from various tissues are maintained <i>in vitro</i> in a nutrient medium for 24 hrs or longer	Cell morphology Viability Proliferation

tissue and organ formation (organogenesis). Such *in vitro* organogenesis models may prove to be valuable to predict the *in vivo* developmental defects (such as deformities in newborns) caused by chemical insults. We have recently investigated the validity of one such system that employs the aggregation of dissociated retinal cells (Figure 6). Individual, isolated, chick-embryo, retinal cells form histotypic aggregates in 24 hours. The toxicity of methylmercury and mercuric chloride (these chemicals cause potential birth defects involving nervous system and sensory organs) was investigated. The results correlated well with those reported on an *in vivo* basis. Kleinschuster et al., (1983) reported that the protective effects of vitamin E and selenium on methylmercury toxicity in this cell aggregation model were similar to those reported in experimental animals. The production of enzyme glutamine synthetase is considered a unique characteristic of the development of the neural retina. This production was inhibited by chemicals that prevented aggregate formation by dissociated cells (Yoneyama et al. 1983).

Summary

Although the *in vitro* models employing cell or tissue cultures promise valuable alternates to the use of live animals in toxicological research, caution must be exercised in relating the *in vitro* data to the intact whole body system. Nevertheless, *in vitro* systems (such as those used at USU) provide an excellent opportunity to predict certain toxic responses and study the underlying biochemical mechanisms. Investigators at Utah State University are continuing to develop and evaluate such *in vitro* models as ways to assess the toxicity of various agricultural and environmental chemicals.

FIGURE 4. Effect of sodium orthovanadate on MDBK cells. (a) untreated cells (b) cells treated with 5×10^{-4} M Na_3VO_4 . Phase contract micrographs ($\times 250$). Note the spindle shaped morphology of treated cells.

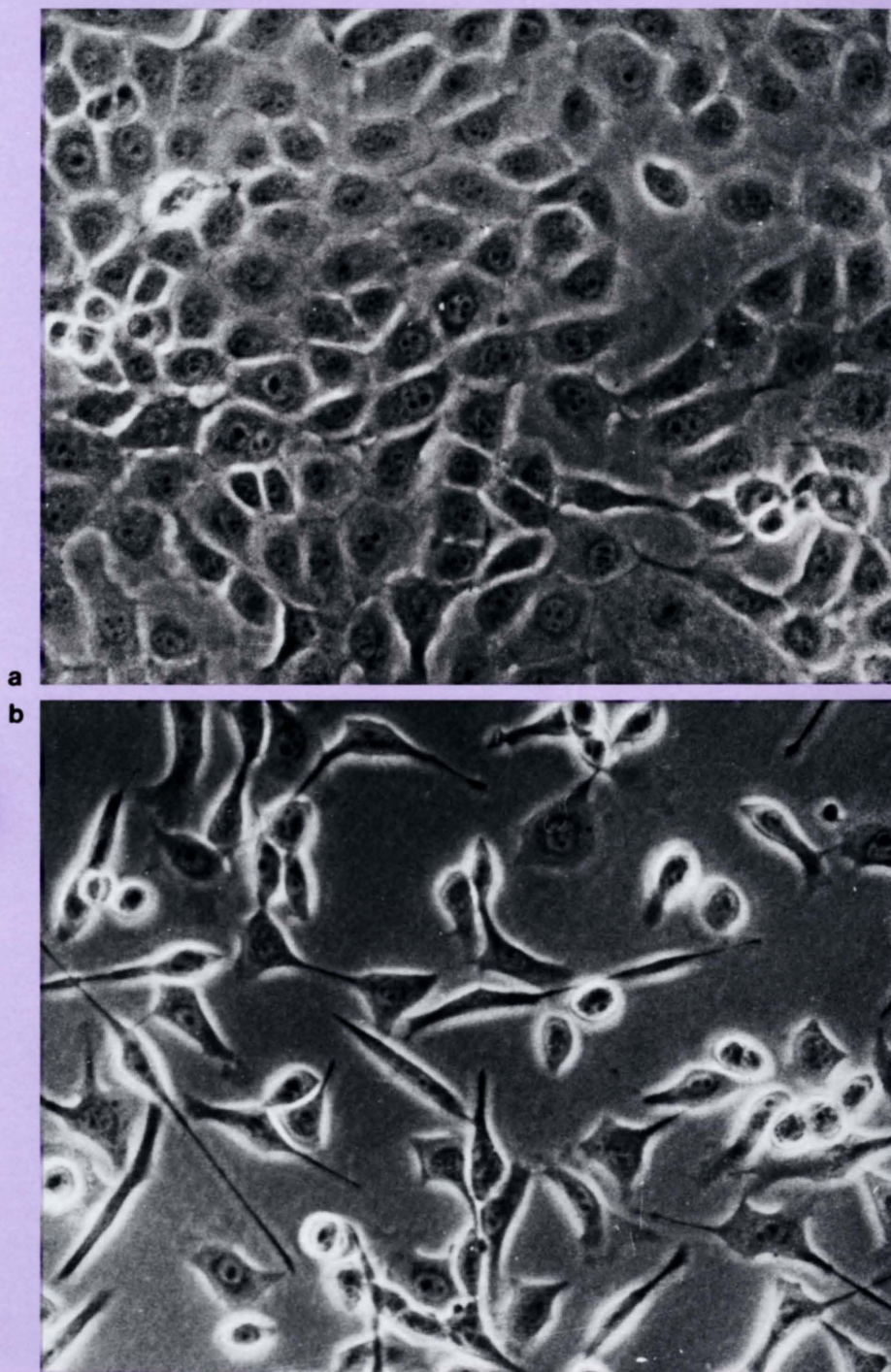
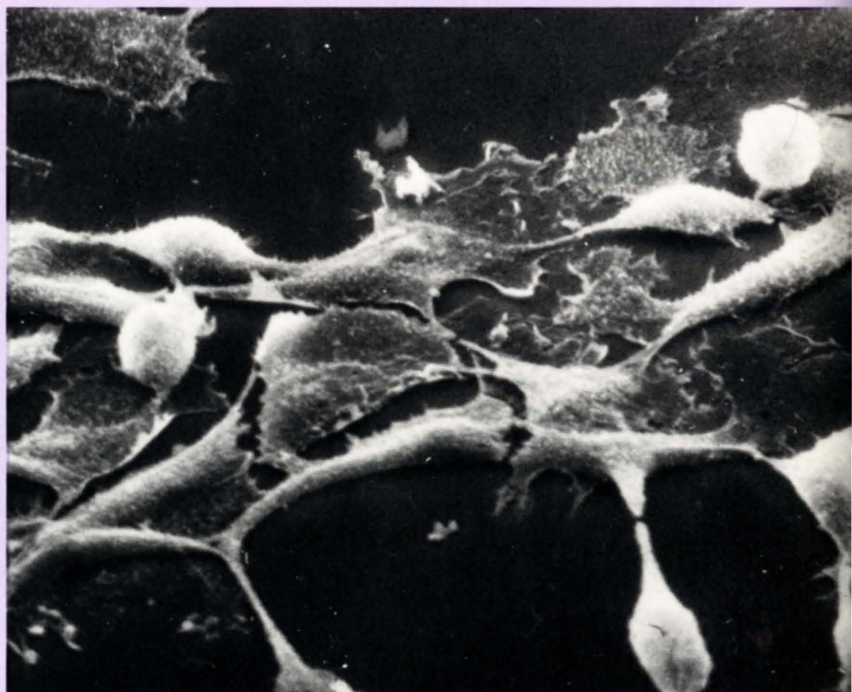
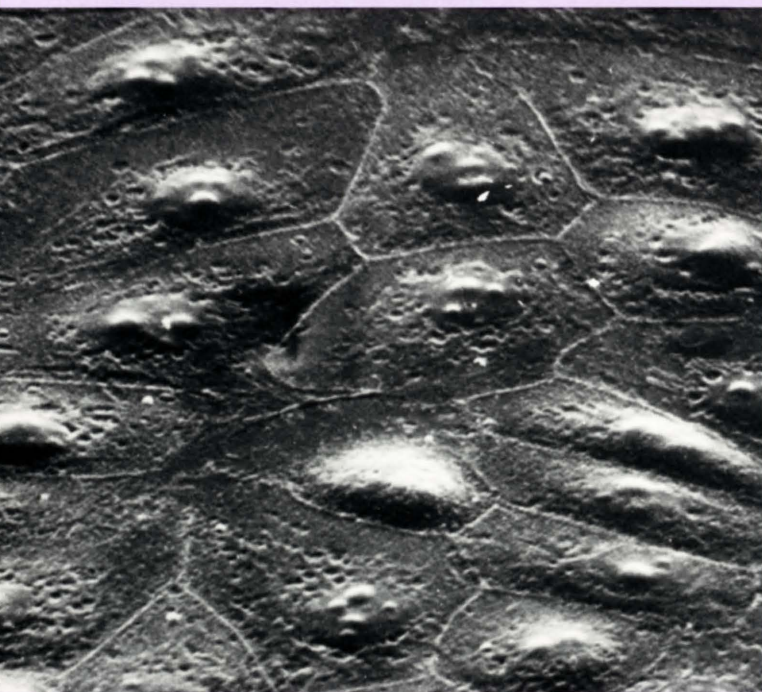
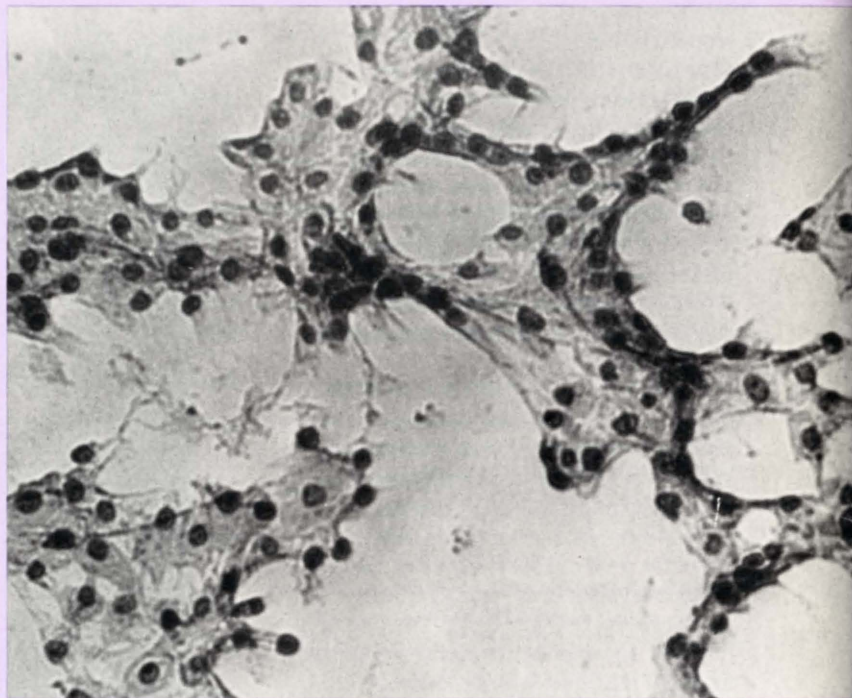
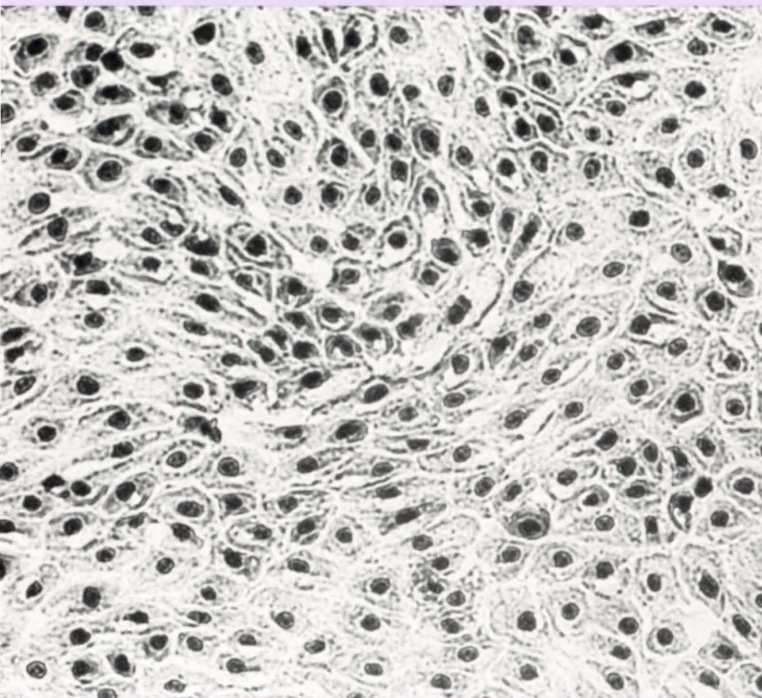
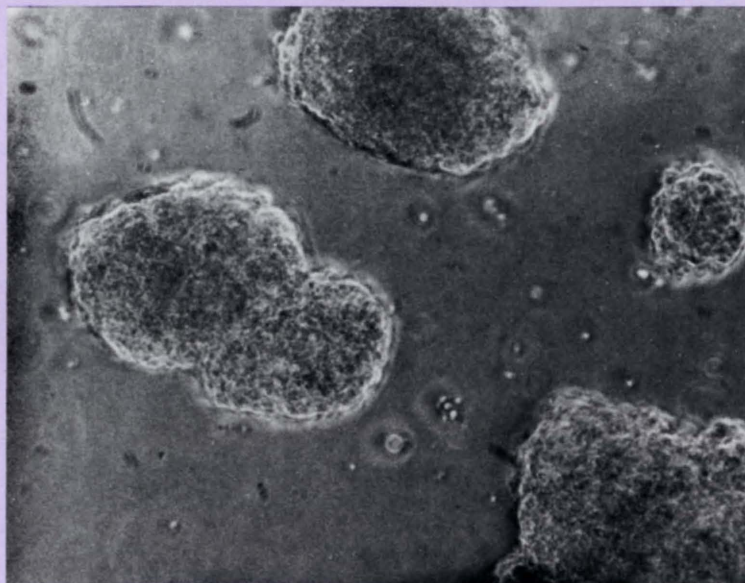


FIGURE 5. Effects of mycotoxins on MDBK cells. (a) untreated MDBK cells, stained by hematoxylin-eosin ($\times 50$). (b) cultures similar to those in (a), but treated with citrinin (a fungal metabolite) at 1×10^{-4} M citrinin for 24 hours ($\times 50$). (c) scanning electron micrograph of untreated cells ($\times 3000$), and (d) scanning electron micrograph of cells treated with 10^{-4} M citrinin for 24 hours ($\times 1500$). Note the lack of surface to surface contact and loss of cytoplasmic materials.

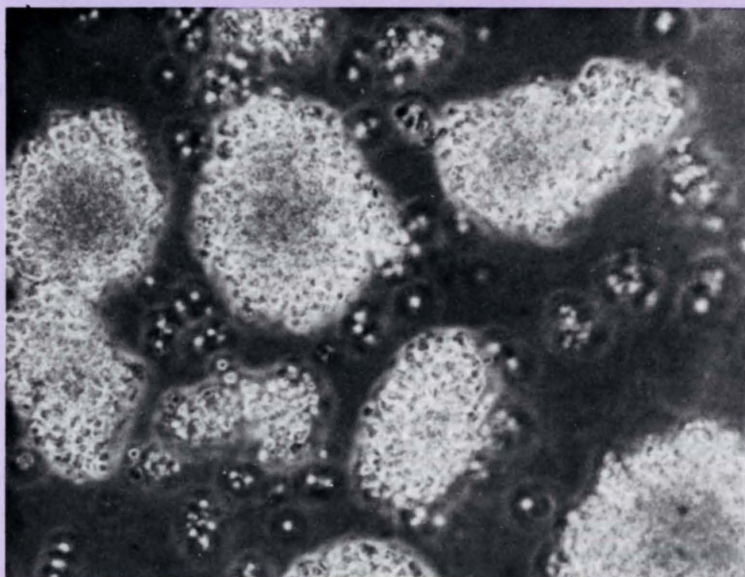
a b



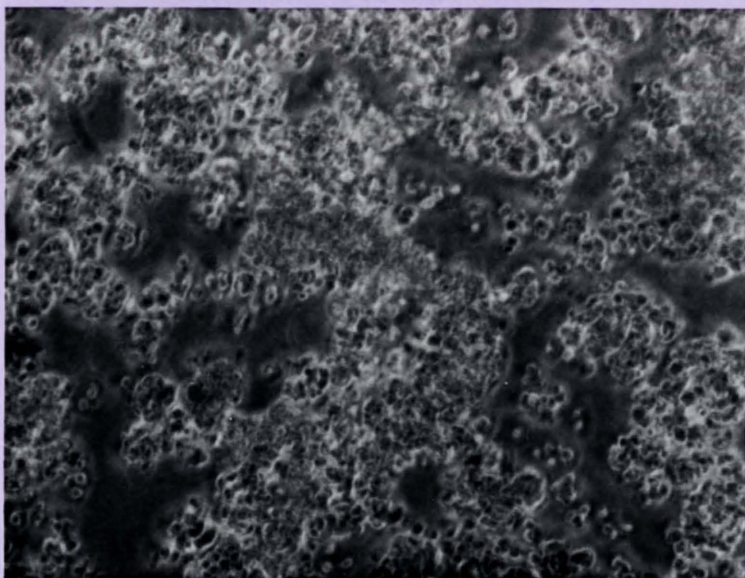
c d



a



b



c

FIGURE 6. Phase contrast micrograph of (a) untreated embryonic chick retinal cell aggregates ($\times 160$). (b) aggregates treated with methylmercury at 10^{-6} M for 24 hours in rotational cultures, (c) aggregates treated with 5×10^{-6} M methylmercury in similar cultures.

REFERENCES

- Bracken, W. M., and Sharma, R. P. (1983). Comparative cytotoxicity and cellular accumulation of HgCl_2 , CdCl_2 and Nd_3VO_4 in bovine kidney cell cultures. *Toxicol Lett.* 18 (Suppl 1):25.
- Kleinschuster, S. J., Yoneyama, M., and Sharma, R. P. (1983). A cell aggregation model for the protective effect of selenium and vitamin E on methylmercury toxicity. *Toxicology* 26:1-9.
- Rowan, A. W. (1979). Alternatives to laboratory animals. The Institute for the Study of Animal Problems. Washington, D.C., 26 pp.
- Sharma, R. P., and Obersteiner, E. J. (1981a). Cytotoxic responses of selected insecticides in chick ganglia cultures. *Can. J. Comp. Med.* 45:60-69.
- Sharma, R. P., and Obersteiner, E. J. (1981b). Metals and neurotoxic effects: Cytotoxicity of selected metallic compounds on chick ganglia cultures. *J. Comp. Path.* 91:235-244.
- Watanabe, P. G., and Sharma, R. P. (1975). Neurotoxicity of organophosphates: Effects of tri-o-tolyl phosphate in chick ganglia cell cultures. *J. Comp. Path.* 85:373-381.
- Yoneyama, M., Sharma, R. P., and Kleinschuster, S. J. (1983). Methylmercury and organogenesis *in vitro*: Inhibition of glutamine synthetase induction and alteration of selected cellular enzymes in aggregation of dissociated embryonic chick retinal cells. *Arch. Environ. Contam. Toxicol.* 12:157-162.

ABOUT THE AUTHORS

Raghubir P. Sharma received his degree in veterinary medicine in India in 1959 and PhD from the University of Minnesota in 1968. Since 1969 he is on the faculty of Utah State University where currently he is professor of toxicology and the chairman of the interdepartmental toxicology program. His research interests include cellular and biochemical toxicology involving chemical effects on nervous and immune systems.

Masao Yoneyama received his MS in toxicology from Utah State University in 1982 and is a graduate research assistant in the toxicology program, working for his PhD. His research areas have been the evaluation of chick retinal aggregation system and mycotoxin effects on bovine kidney cells.

INDEX FOR 1984

R. S. Albrechtsen

New Grain Varieties for Utah
45(1):14-21 (joint author)

C. Amrhein

The Sodic Hazard in Coal Mine
Overburden
45(1):28-31 (joint author)

J. O. Anderson

A Feed Source of the Future
45(4):114-119 (joint author)

D. C. Aston

Reducing Greenhouse Energy
Requirements
45(3):87-88 (joint author)

D. Bailey

Fat Cattle: Regional Price Differences
1978-1983
45(4):100-103 (joint author)

B. B. Barnett

The Malnutrition/Diarrhea Connection
45(4):104-109 (joint author)

D. T. Bartholomew

Mutton in Summer Sausage
45(4):124-127 (joint author)

J. C. Batty

Reducing Greenhouse Energy
Requirements
45(3):87-88 (joint author)

J. L. Blake

Phosphorus Needs of Beef Cattle
45(4):97-99 (joint author)

W. A. Brindley

Unity Against Resistance
45(2):59-61

B. W. Brorsen

Fat Cattle: Regional Price Differences
1978-1983
45(4):100-103 (joint author)

A. Brown

The Sodic Hazard in Coal Mine
Overburden
45(1):28-31 (joint author)

L. M. Brown

Tracking the Sunflower Bees
45(2):41-43 (joint author)

J. E. Butcher

Phosphorus Needs of Beef Cattle
45(4):97-99 (joint author)

M. D. Butler

Reducing Greenhouse Energy
Requirements
45(3):87-88 (joint author)

J. W. Call

Phosphorus Needs of Beef Cattle
45(4):97-99 (joint author)

W. F. Campbell

Tough Turfs for Utah
45(2):36-40 (joint author)

R. L. Cartee

Precipitation vs. Wheat Yields
45(3):66-67 (joint author)

T. D. Chaar

The State of the State Climatology Office
45(2):44-47

B. Chesler

The Little Sahara
45(2):48-52 (joint author)

H. M. Deer

Pesticide Programs at USU
45(1):4-7

W. G. Dewey

New Grain Varieties for Utah
45(1):14-21

D. C. Dobson

A Feed Source of the Future
45(4):114-119 (joint author)

P. Dryden

Pinto Bean Root Rot
45(1):57-58 (joint author)

Y. Elsner

Comparable Bone Defects
45(4):110-113 (joint author)

J. O. Evans

Goatsrue Eradication
45(1):8-11 (joint author)

E. J. Gardner

Comparable Bone Defects
45(4):110-113 (joint author)

J. Giannini

Effects of Excessive Fluoride
45(3):89-95 (joint author)

L. F. Hall

The Little Sahara
45(2):48-52 (joint author)

R. G. Hansen

Pantothenic Acid in the Human Diet
45(1):120-123 (joint author)

J. J. Jurinak

The Sodic Hazard in Coal Mine
Overburden
45(1):28-31 (joint author)

N. C. Leone

Comparable Bone Defects
45(4):110-113 (joint author)

A. W. Mahoney

The Malnutrition/Diarrhea Connection
45(4):104-109 (joint author)

J. Manwaring

Effects of Excessive Fluoride
45(3):89-95 (joint author)

V. T. Mendenhall

The Art of Home Canning
45(3):68-72

G. W. Miller

Effects of Excessive Fluoride
45(3):89-95 (joint author)

H. Y. Nam

Elderly Migration Patterns in Utah
45(3):63-65 (joint author)

R. F. Nielson

Precipitation vs. Wheat Yields
45(3):66-67 (joint author)

A. E. Olson

Phosphorus Needs in Beef Cattle
45(4):110-113 (joint author)

Comparable Bone Defects
45(4):110-113 (joint author)

C. I. Osula

Mutton in Summer Sausage
45(4):124-127 (joint author)

NEW PUBLICATIONS 1984

F. D. Parker

Tracking the Sunflower Bees
45(2):41-43 (joint author)

F. D. Provenza

Interseeding Crested Wheatgrass Ranges
45(3):73-77 (joint author)

J. C. Pushnik

Effects of Excessive Flouride
45(3):89-95 (joint author)

J. H. Richards

Interseeding Crested Wheatgrass Ranges
45(3):73-77 (joint author)

M. D. Rumbaugh

Legumes for Wildland Plantings
45(1):22-27

R. P. Sharma

In Vitro Toxicity Evaluation
45(4):128-131 (joint author)

L. M. Shultz

Crownvetch
45(1):12-13

J. L. Shupe

Phosphorus Needs of Beef Cattle
45(4):97-99 (joint author)
Comparable Bone Defects
45(4):110-113 (joint author)

R. W. Sidwell

The Malnutrition/Diarrhea Connection
45(4):104-109 (joint author)

W. O. Song

Pantothenic Acid in the Human Diet
45(4):120-123 (joint author)

A. R. Southard

The Little Sahara
45(2):48-52 (joint author)

N. J. Stenquist

A Feed Source of the Future
45(4):114-119 (joint author)

W. F. Stinner

Elderly Migration Patterns in Utah
45(3):63-65 (joint author)

J. Y. Takemoto

Basic Research in Photosynthesis
45(1):1-3

N. Van Alfen

Pinto Bean Root Rot
45(2):57-58 (joint author)

W. A. Varga

Tough Turfs for Utah
45(2):36-40 (joint author)

D. R. Walker

Reducing Greenhouse Energy
Requirements
45(3):87-88 (joint author)

L. S. Willardson

Drain Envelopes
45(2):33-35

B. L. Woodbury

Mutton in Summer Sausage
45(4):124-127 (joint author)

B. W. Wyse

Pantothenic Acid in the Human Diet
45(4):120-123 (joint author)

M. Yoneyama

In Vitro Toxicity Evaluation
45(4):128-131 (joint author)

S. A. Young

Seed Certification in Utah
45(3):78-86

N. N. Youssef

The Chalk Brood Syndrome in Wild Bees
45(2):53-56

Publications released by the Utah Agricultural Experiment Station in 1984 are listed at right. For a free copy of any issue outlined, or to have your name placed on our mailing list for a Catalog of Publications, or to receive UTAH SCIENCE Magazine, please send your name, request, and mailing address to: UAES Publications Office, UMC 48, Utah State University, Logan, Utah 84322.

NUMBER

TITLE

RESEARCH REPORTS

- 92** ENVIRONMENTAL FACTORS ASSOCIATED WITH YIELD DIFFERENCES BETWEEN SEEDING DATES OF SPRING WHEAT
B. S. Sharratt, R. J. Hanks and J. K. Aase
- 93** GROWTH TRENDS IN ELDERLY POPULATIONS IN METROPOLITAN AND NONMETROPOLITAN UTAH: 1950-1980
W. F. Stinner, L. Baal and H. Y. Kwon
- 94** 1983 SMALL GRAIN PERFORMANCE TRIALS IN UTAH
R. S. Albrechtsen and W. G. Dewey
- 95** THE UTAH STATE UNIVERSITY LACTIC-CULTURE SYSTEM UPDATE
G. H. Richardson and C. A. Ernstrom
- 96** 1983 TURKEY RESEARCH PROJECTS PROGRESS REPORT
R. E. Warnick
- 97** USE OF SALINE WASTE WATER FROM ELECTRICAL POWER PLANTS FOR IRRIGATION 1983 REPORT
R. J. Hanks, R. F. Neilson, R. L. Cartee, L. S. Willardson, R. E. Long and R. B. Sorensen

RESEARCH BULLETINS

- 508** MATHEMATICAL MODELS FOR ESTIMATING ENERGY AND PROTEIN UTILIZATION OF FEEDSTUFFS
M. F. Wardeh, P. V. Fonnesebeck, and L. E. Harris

SPECIAL REPORT

- 24** IMPORTANT FARMLANDS OF WASHINGTON COUNTY
A. R. Southard

BOOKLET

GROUND COVERS
W. A. Varga

UTAH SCIENCE

UTAH AGRICULTURAL EXPERIMENT STATION
UTAH STATE UNIVERSITY, UMC 48
LOGAN, UTAH 84322

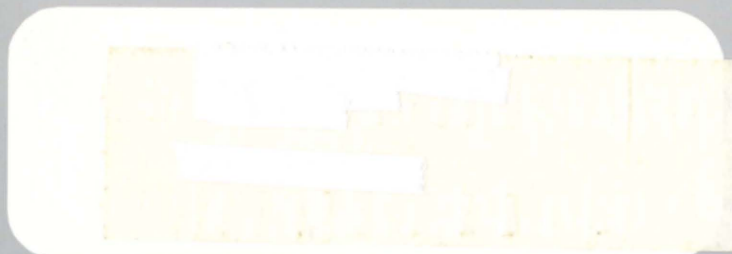
Doyle J. Matthews
DIRECTOR

Address Correction Requested



BULK RATE

POSTAGE & FEES
PAID AGRI 101
PERMIT NO. G 269



UTAH SCIENCE is a quarterly magazine devoted primarily to the report of experiment station research in agriculture and related fields of study (i.e., crops, land and water resources, animal/dairy management, forestry, home and community life, human nutrition and development). The purpose of the magazine is to offer a tangible and usable representation of the expenditure of tax dollars, that will unite the public with the experiment station through shared research results. Published by the Utah Agricultural Experiment Station, Utah State University, UMC 48, Logan, Utah 84322.

The magazine will be sent free on request. Please include a mailing label from a recent issue of UTAH SCIENCE with any request for change of address.

To avoid overuse of technical terms, sometimes trade names of products or equipment are used. No endorsement of specific products or firms named is intended, nor is criticism implied of those not mentioned.

Articles and information appearing in UTAH SCIENCE become public property upon publication. They may be reprinted provided that no endorsement of a specific commercial product or firm is stated or implied in so doing. Please credit the authors, Utah State University, and UTAH SCIENCE.

Stanford Cazier
PRESIDENT
UTAH STATE UNIVERSITY

Doyle J. Matthews
DIRECTOR
AGRICULTURAL EXPERIMENT STATION

C. Elmer Clark
ASSOCIATE DIRECTOR
AGRICULTURAL EXPERIMENT STATION

Thaya Davis Chaar
ACTING EDITOR

Carol Rae Grundmann
ART DIRECTOR

Utah State University is committed to a policy of equal opportunity in student admission, student financial assistance, and faculty and staff employment and advancement, without regard to race, color, religion, sex, age, national origin, or handicap.